NEW SERIES, VOL. XL JUNE, 1938 No. 3

The American Journal of Surgery

is the leading independent surgical Journal. It publishes many papers read before the outstanding Surgical Societies, but it is *not* "the official organ" of any organization.

Copyright, 1938, by The American Journal of Surgery, Inc.

EDITORIAL BOARD

Editor:

Thurston Scott Welton

New York

D. C. Balfour *Rochester, Minn.*
Carl Beck *Chicago*
George R. Brighton *New York*
Meredith F. Campbell *New York*
James T. Case *Chicago*
Isidore Cohn *New Orleans*
Bradley L. Coley *New York*
F. A. Coller *Ann Arbor*
George W. Crile *Cleveland*
E. C. Cutler *Boston*
R. V. Day *Los Angeles*
Charles A. Elsberg *New York*
Herbert C. Fett *Brooklyn*
C. R. G. Forrester *Chicago*
H. Dawson Furniss *New York*
J. H. Gibbon *Philadelphia*
Emil Goetsch *Brooklyn*
Charles A. Gordon *Brooklyn*
Donald Guthrie *Sayre, Pa.*
A. E. Hertzler *Kansas City*
Louis J. Hirschman *Detroit*
James M. Hitzrot *New York*
Frederick C. Holden *New York*
Emile F. Holman *San Francisco*
J. A. Hyams *New York*
R. H. Jackson *Madison*
John E. Jennings *Brooklyn*
W. L. Keller *Washington, D.C.*
H. A. Kelly *Baltimore*
T. J. Kirwin *New York*
Arthur Krida *New York*
A. V. S. Lambert *New York*
Maurice Lenz *New York*
H. H. M. Lyle *New York*
Jerome M. Lynch *New York*
Urban Maes *New Orleans*
Harrison S. Martland *Newark*
Rudolph Matas *New Orleans*
Roy D. McClure *Detroit*
H. C. Naffziger *San Francisco*
C. R. O'Crowley *Newark*
Louis E. Phaneuf *Boston*
Eugene H. Pool *New York*
Douglas Quick *New York*
N. P. Rathbun *Brooklyn*
H. A. Royster *Raleigh*
Henry S. Ruth *Philadelphia*
A. C. Scott *Temple*
M. G. Seelig *St. Louis*
J. Bentley Squier *New York*
H. J. Stander *New York*
George W. Swift *Seattle*
H. L. Updegraff *Los Angeles*
Grant E. Ward *Baltimore*
J. H. Woolsey *San Francisco*

EDITORIAL

ORGANIZED MEDICINE

SHORTLY after this issue of the Journal appears, the annual convention of the American Medical Association will be over. Most state meetings for 1938 will have become history. Legislatures will go home and legislative matters will lie dormant until the fall. During the summer most of us will be more concerned with vacations than with economic problems, although the economic problems will not vanish with our lapse of attention.

As the summer dies, we shall most probably begin again where we left off, and doubtless we shall again be engulfed by countless proposals for the salvation of our calling. This seems to have been the pattern for some time past, and we shall no doubt continue according to form.

No one will deny that we are today confronted with many major questions—questions which organized medicine must (and undoubtedly one day will) solve. By organized medicine we mean that body of about one hundred thousand physicians who, through constituent county and state societies, make up the American Medical Association. Economic and social problems are discussed at the meetings of the county societies, who send delegates, instructed on the major issues, to the state meetings. The latter, after appropriate discussion, send their instructed delegates to the sessions of the House of Delegates of the Association. This is the deliberate, roundabout, but sure method by which organized medicine functions.

It is therefore necessary in order to tackle the problems of the profession, to consider them at meetings of the county societies. After debate, committee study and reports, and membership discussion, conclusions may be arrived at by the majority. Should other counties agree with the decisions, resolutions of the State societies are next in order. Finally, these economic and social problems properly reach the

parent body, the Association itself, and come up for discussion and possible solution.

For the rank and file of the profession to abide by the findings of the legislative body of the A.M.A. is the one possible course. If one does not agree with these findings, he should voice his disapproval at the meetings of his county society. If his arguments have great merit and weight, members of other county societies will join his dissent; the state societies in turn will join the chorus. Finally, if enough states go on record, the matter will be righted at a future meeting of the A.M.A.

There is no place in American medicine for any but the duly elected members of the county societies and those chosen to speak for them. Self-appointed outsiders, or special groups of physicians, who offer resolutions or proposals aimed at solving pertinent problems confronting the profession may do great harm through their activities outside the organized field. No doubt most of these men (many of them research workers, full time teachers, who never have practiced medicine, or men who have never joined properly accredited medical societies) are completely sincere in the belief that they are following proper aims along proper channels. But the majority of the physicians of the country feel that they are out of order and that their indiscriminate efforts, if taken seriously, will inevitably result in chaos and disorder.

This is a plea for physicians to become members of organized medicine and to discuss and settle medical problems within the fold. Only in unity is there strength, and these days the medical profession needs all the strength it can muster. The diverging statements of groups and outside factions can only confuse the public. The dissenting associations may appear to represent the majority of American physicians, when, as a matter of fact, compared with the thousands in the organized A.M.A., they are only a tiny minority. The politicians, reasoning that medicine is composed of divided, unorganized cliques without weight or power, will feel free to rush through legislation of possibly harmful effect.

Secession from the parent organization is unthinkable; it spells disaster and doom. Independent groups edging into the center of the public spotlight put organized medicine into a false position before the lay public. If the members of the independent groups are truly sincere in their efforts to reach a medical Utopia, they will spend their energies to better advantage within the legal confines of organized medicine.

T. S. W.

CORRECTION

In the article on Pyogenic Abscess of the Liver by Dr. Alton Ochsner, Dr. Michael DeBakey and Dr. Samuel Murray, which appeared in the April 1938 issue, the mortality rate in a collected series of ninety-seven cases of pyogenic liver abscess with complications was given incorrectly as 20.7 per cent. This should have read 90.7 per cent.

Subscribers to The American Journal of Surgery *visiting New York City are invited to make the office of the publishers (The American Journal of Surgery, Inc., 49 West 45th Street, New York) their headquarters. Mail, packages or bundles may be addressed in our care. Hotel reservations will gladly be made for those advising us in advance; kindly notify us in detail as to requirements and prices.*

ORIGINAL ARTICLES

INTESTINAL OBSTRUCTION*

AN ANALYSIS OF 200 CASES ATTEMPTING IMPROVED CORRELATION OF MORTALITY, PATHOLOGIC PHYSIOLOGY, AND SIGNS AND SYMPTOMS

J. K. DONALDSON, M.D.

Associate Professor of Surgery, University of Arkansas Medical School

LITTLE ROCK, ARKANSAS

IN a recent discussion of intestinal obstruction the author stressed various morbid processes of the disease as they occur in different conditions ordinarily listed under the intestinal obstruction heading, and presented a simplified classification based upon pathologic physiology.[1] The chief efforts made in this present article are to analyze mortality rates and signs and symptoms in different types of obstruction, and to appreciate more fully the distinction between *early* and *late* signs as they are interpreted in the light of previously discussed pathologic fundamentals.[1]

In many textbooks of surgery one finds numerous diagnostic signs and symptoms listed, with insufficient stress on the fact that many of these may frequently occur only at advanced stages in the morbid process, and that they are somewhat variable in different types of obstructions. McIver, in 1933, in speaking of morbid processes occurring in different types stressed the fact that they are often at wide variance with each other: "This lack of uniformity in clinical picture, etiology and pathology creates a situation which is complicated and lends itself to misunderstanding and confusion, more especially since in spite of its very broad meaning the term intestinal obstruction is often used as though it represented a single clear-cut entity."[4]

Of course it is impractical for brief surgical texts to attempt undue detail. Nevertheless, those relying upon textbook descriptions of intestinal obstruction should appreciate their limitations, since inadequate criteria may become dangerous. This would seem true, for example, concerning distention. Nearly all the texts describe or allow the assumption that distention is an early part of the picture of obstruction when actually it may not be at all. The following are excerpts from popular books. "Distention from gas (tympanites) is always soon present." "Distention of the abdomen usually occurs—." Another speaks of "gaseous distention of the bowel, without abdominal rigidity," and again, "meteorism—gaseous distention of the intestine—" without any correlation with pathologic stages.

However, statistics show that even in simple obstructions the mortality is very high if one waits more than forty-eight hours from the onset of obstruction to institute proper treatment, not to speak of the necessity for still earlier treatment in gangrenous and folded loop cases. It is therefore easy to see why it behooves us to know just what signs and symptoms we may depend upon to diagnose a given case in a truly early stage.

Because of inadequate detail in the charts studied, only fifty-one of the two hundred reviewed were incorporated for

* From the Department of Surgery, University of Arkansas Medical School, Little Rock, Arkansas.

final analysis, and deficiencies are present in some of these.* We were able, however, to designate with a satisfactory degree of accuracy distention as it occurred in twelve-hour period groups as measured from time of onset in a number of cases. Deep seated tenderness, constipation, pain and vomiting were noted fairly consistently. No other signs or symptoms were found to be or considered as of definite importance for the purpose of this analysis, though in so far as was possible an attempt was made to list them. *X*-ray findings were not incorporated.

MORTALITY IN DIFFERENT TYPES AND RELATIONSHIP OF THE TIME FACTOR†

No effort will made here to review in detail all the facts in the tables, but a number of findings warrant comment:

The total mortality for all the cases in the tables was 58.8 per cent. That the folded loop obstructions are more serious than the simple ones is illustrated in that the mortality for the former was approximately 12 per cent higher (71.4 per cent) than the latter (59 per cent) despite the fact that cases with folded loop obstructions were operated on approximately twenty-two hours earlier from onset (average 37.45 hours) than the simple ones (average 59.5 hours). When even simple obstructions went over forty-eight hours from onset without proper treatment the mortality rate was very high (84.6 per cent); and when over seventy-two hours the mortality was 100 per cent. When operation was done at or under forty-eight hours (average 32 hours) in this series the mortality was 12.5 per cent.

A mortality rate below the average for obstructions was present with obstructing hernias (27.2 per cent). This was probably due to the previous education of the patient, the earlier diagnosis and earlier operation (average 30 hours).

The 100 per cent mortality in the two cases of mesenteric thrombosis and the 50 per cent mortality in the three intussusception cases are roughly comparable with mortality statistics of others.

The seriousness of cases with gangrene was demonstrated by a 100 per cent mortality, a figure higher than in some other series. Only one patient with gangrene was operated on within twelve hours from onset and he died. One may evidently assume that we must operate on such patients within twelve hours if a reasonably low mortality is to be obtained. Absorption of toxin probably begins within thirty minutes after arterial blood supply is completely blocked.[3]

SIGNS AND SYMPTOMS IN DIFFERENT TYPES: FREQUENCY AND TIME OF DEVELOPMENT

Table v was compiled in such a way that I believe it to be a reasonably accurate index to the time in which *distention* occurs in different types. In the simple obstructions, in no instance was distention definitely noted in the first twenty-four hours. It was definitely observed that it was not present in seven and four of these cases in the first and second twelve-hour periods respectively. In the third twelve-hour period, up to and including the thirty-sixth hour, the chances as calculated here seem four to one that clinical distention of note will not be present in simple obstructions. The writer would not be justified in stipulating that in the fourth twelve-hour period, simple obstruction would present clinical distention in only 50 per cent of the cases, as is indicated in this series; but he can say definitely that he has seen many cases which did not present clinical distention in this length of time, and this 50 per cent figure would seem reasonably accurate. After forty-eight hours nearly all cases of simple obstruction will present clinical distention, but it may not be marked in some in the earlier part of the fifth twelve-hour period.

* The case records were obtained from the Little Rock General Hospital and St. Vincent's Infirmary. The writer wishes to acknowledge the courtesy of Sister Teresa, Mrs. Loula R. Ashbaugh and Miss Helen Robinson in collection of the histories.

† Cases in which operation was markedly delayed were excluded in calculation of the following averages.

TABLE I

SIMPLE OBSTRUCTIONS

Case Number	No. Hrs. before Operated	Distention	Vomiting	Gangrene	Remarks	Result
132335	7 days	3 days	?	No	No B.M. 7 days	Died
18548	6 days	Eventually	Early	No	Probably partial obstipation for a time.	Died
233735	4 days (?)	At 3 days (?)	Early	No	No B.M.	Died
18971	Died fourth day without surgery	?	?	?	No B.M.	Died
18582	Not operated. Died after fourth day	At 3 days	Within 1 hr.	No	No undue rigidity but tense. One B.M. since onset. No masses.	Died
1321	96 hrs.	Slight at 48	3 hrs.	No	Slight rigidity and moderate tenderness. Visible peristalsis at 48 hrs.	Died
23234	90 hrs.	Marked at 90	?	?	Small B.M. twice after onset. No masses.	Died
19438	84 hrs.	84 hrs.	Yes	?	Tender and tense at 84.	Died
23161	80 hrs.	None at 12 hrs. Some at 48 hrs. Definite at 72 hrs.	None at 24 hrs. Once at 48 hrs.	No	Enema slight results once. No rigidity 12 hrs. Moderate diffuse at 12 hrs. Abdomen silent at 12.	Died
1670	72 hrs.	72 hrs.	Onset	No	Tender at 72 hrs. No definite rigidity.	Died
1504	70 hrs.	"Somewhat" at 48	?	No	Slight tenderness at 48 hrs. No masses.	Died
19438	50 hrs.	Marked at 50 hrs.	At onset	No	Rigidity definite at 50 hrs. No B.M.	Died
24060	40 hrs.	At 36 hrs.	?	No	No B.M. Enema no results.	Died
251935	70 hrs.	At 36 hrs.	Early	No	Slight B.M. nausea and successful duodenal drainage 36 hrs.	Lived
21916	50 hrs.	No distention at 18 hrs.	6½ hrs.	No	Some general tenderness and local rigidity at 18 hrs.	Lived

TABLE I (*Continued*)

Case Number	No. Hrs. before Operated	Distention	Vomiting	Gangrene	Remarks	Result
102535	48 hrs. ?	?	Continuous	No	No B.M. Enema results.	Lived
126635	48 hrs.	?	None at 10 hrs.	No	.....	Lived
4141	48 hrs.	No distention clinically but marked at table.	?	Dark splotches present on gut.	No B.M.	Lived
23406	36 hrs.	Not marked at 36 hrs.	Moderately early	No	.....	Lived
22594	At 18 hrs.	"Moderate" tympany at 18 hrs. Enema clear	In 4–5 hrs.	No	.....	Lived
65035	10 hrs.	None at 10 hrs.	None at 10 hrs.	No	.....	Lived
260136	6 to 10 hrs.	None	Apparently not	No	.....	Lived

Average time operated from onset excluding abnormally delayed cases—59.5 hrs.
Total died..... 13
Total lived..... 9
Mortality..... 59 per cent

It should be said that when distention of the abdomen is not present at inspection, the coils of intestine at the operating table may, nevertheless, possibly be found distended, to the extent that toxin absorption is evidently occurring. One may appreciate the importance of fully realizing that distention actually is a late sign by correlating the time of occurrence of distention with the mortality statistics which are presented here and elsewhere. Since distention in simple obstructions does not seem to occur with any great degree of consistency until the fourth twelve-hour period, and since it has been repeatedly demonstrated that the mortality is unduly high unless relief is given before forty-eight hours have passed, *we must consider distention as usually a late sign.*

Table v shows that distention in folded loop obstructions in this series seemed to come earlier than it did in simple obstructions. Correlating this with the time and mortality factor as listed in Table II, one may see that in folded loop obstructions lethal factors function earlier and more rapidly than in simple ones. Therefore, distention, though coming withing a few hours from onset of obstruction, nevertheless may appear only late in the morbid picture; and again one should surely not depend upon it as an early diagnostic sign.

It is seen that distention in strangulated hernias would seem to occur about as in simple obstructions. Proper interpretation of the pathologic physiology of this type of obstruction would lead one to expect this.

In mesenteric thrombosis, intussusception and other conditions in which gangrene is present, one surely must not wait for distention to develop, since gangrenous tissue predestines death within a very few hours.

Perusal of the tables demonstrates the fact that *vomiting* is a fairly constant symptom, occurring early in the majority

TABLE II

FOLDED LOOP OBSTRUCTIONS

Case Number	No. Hrs. before Operated	Distention	Vomiting	Gangrene	Remarks	Result
3037	6 days	?	Yes	Yes	?	Died
185735	72 hrs.	At 72 hrs.	Onset	No ? Volvulus	Abdomen silent on admittance.	Died
22290	55 hrs.	Slight at 48 hrs.	Prompt	No	No B.M. (some muscle tension r.l.q. and marked tenderness r.l.q. at 48 hrs. Sharp onset.	Died
22594	50 hrs.	At 18 hrs.	4–5 hrs.	Yes	Enema no results. Low ileum obstipation.	Died
22984	50 hrs.	Presume not at 50 hrs.	At 12 hrs.	Yes	Moderate rigidity, tenderness, 26 hrs.; peristalsis visible 50 hrs.	Died
177034	Died at 50 hrs. without surgery.	Not marked at 48 hrs.	Early	Yes	Rather generalized mod. spasm and tenderness.	Died
176134	38 hrs.	Not marked if present at 36 hrs.	Early	No	"Slight muscle spasm and tenderness" at 36 hrs.	Died
1047	36 hrs.	Very slight 12 hrs.	Prompt	Yes	No B.M. Enema no results. Mass palpable at 12 hrs. Moderate tenderness to deep pressure.	Died
22690	24 hrs. plus	Marked at 24 hrs.	?	Yes. Volvulus of sigmoid	Abdomen silent. Diffuse tenderness. Enema no results; no B.M.	Died
25041	12 hrs.	At 12 hrs.	?	Yes. Large volvulus	Rigid at 7. Diffuse tenderness. Silent at 12 hrs.	Died
4177	48 hrs.	Moderate at 18 hrs. More def. at 48 hrs.	At onset	Hemorrhagic, but believe not gangrenous. Resected 2 ft.	Diff. tenderness and muscle spasm 18 hrs. Rigidity and distention more def. at 48 hrs.	Lived
3497	48 hrs.	Moderate 24 hrs.	Prompt	Apparently not, but hemorrhagic infarction.	"Tenderness and some spasm 24 hrs."	Lived
49036	24–36 hrs.	Evidently none at 24–36 hrs.	Early	No	B.M. not mentioned	Lived
3598	10½ hrs.	None at 10 hrs.	None at 10 hrs.	No	No particular nausea. Some increased peristalsis. No b.m. No results to enema 10 hrs.	Lived

Average time operated from onset excluding abnormally delayed cases—37.45 hours.

Total died..... 10

Total lived..... 4

Mortality..... 71.4 per cent

of all cases studied. It may be said, however, that it is not always so continuous and constant as is sometimes stated. It seems to have no markedly distinguishing characteristics in different types, though it will not occur persistently or consistently in purely venous mesenteric occlusions from the records to discover any distinguishing characteristics of the pain in any particular type of obstruction. In a general way, however, it would seem more sharp and severe in the folded loop obstructions than in the simple ones. It has, of course, long been recognized that sudden strangu-

TABLE III

STRANGULATED HERNIA

Case Number	No. Hrs. before Operated	Distention	Vomiting	Gangrene	Remarks	Result
21902	4 days	Not marked at 4 days	In few hours	No	.....	Died
2668	65 hrs.	Flacid 24 hrs.	5 hrs.	Yes. Femoral	No rigidity. B.M. after onset.	Died
23951	48 hrs.	"Somewhat" at 24 hrs.	Early and continuous	No	.....	Died
221334	48 hrs.	Slight at 48 hrs.	Rather constant	No	General tenderness. No rigidity.	Lived
274235	48 hrs. plus	.....	Within 6 hrs.	No	.....	Lived
116537	36 hrs.	No	Early	No	No rigidity.	Lived
23883	22 hrs.	Apparently not	At 10 hrs.	No but darkened	.....	Lived
298635	12–18 hrs.	No	Yes	No	.....	Lived
231136	6–7 hrs.	No	Early	No	.....	Lived
17736	6 hrs.	Apparently not	In few hrs.	No	.....	Lived
23918	5½ hrs.	Apparently not	?	No	.....	Lived

Average time operated from onset excluding abnormally delayed cases—30 hours.

Total died..... 3
Total lived..... 8
Mortality..... 27.2 per cent

without direct lumen obstruction.[2] It is probably more rapid in onset in high simple obstructions, folded loop and strangulated obstructions than in low simple ones. In persistency, severity and frequency it occurs in the different types in the order in which they are named here, i.e., most gravely in high simple obstructions, less so in folded loop and strangulated ones.

Pain at onset is indeed a constant symptom, occurring early in every one of the 200 cases studied. It was impossible lation of arterial blood supply gives sudden severe pain. Pain in purely venous mesenteric thrombosis without direct gut lumen obstruction is somewhat less severe, and is of colicky nature extending over a longer course than in most types of obstruction.[2]

Visible peristalsis does not seem to be at all a constant finding. In only one case of the 200 was there a note that it was present.

The study well bears out the statements repeatedly made that one or two bowel

movements may occur as the bowel below the obstruction is emptied. *Obstipation*, however, in complete mechanical obstructions is certainly a constant and fairly reliable finding, with the exception of these one or two potential movements. But in the purely venous type of mesenteric occlusion without direct gut lumen obstruction, tention may be a lethal factor in obstruction in that it may cause an ischemia of mucous membrane, allowing toxins to be absorbed.

Loss of essential salts and fluids is likely to be more rapidly serious and disturbing in obstructions high up in the small gut than in lower obstructions.

TABLE IV

MESENTERIC THROMBOSIS AND INTUSSUSCEPTION

Case Number	No. Hrs. before Operated	Distention	Vomiting	Gangrene	Remarks	Result
161234	7 days	Def. at 7 days	For duration of illness. No blood seen.	Yes (?) Hemorrhagic infarction.	Constant dull pain one week with pulling pain. B.M. reg. General tenderness and muscle spasm.	Died. Mesenteric thrombosis, evidently venous.
179834	48 hrs. Iliostomy (died)	Marked 48 hrs.	Within few hrs.	Yes	Tender entire abdo. at 48. Intra fluid wave present. App. no fluid levels in gut. Indocarditis.	Died. (Mesen. throm.)
37434	27 hrs.	Slight at 27 hrs.	In 6 hrs.	?	No B.M. from enema and no blood. Slight muscle spasm 6 hrs. Gen. tenderness.	Died. Intuss.
90834	48 hrs.	Evidently somewhat	Onset	No	Movable mass cecal region.	Lived. Intuss. Fibroma small intestine
69335	24 hrs.	?	Prompt	No	Bloody stool to enema.	Lived. Intuss.

bowel movements may occur daily over many days, as happened in the one (apparent) case of this series.

REVIEW OF PATHOLOGIC PHYSIOLOGY AND DIVISION OF SIGNS INTO EARLY AND LATE ONES

One should keep in mind that interference with the arterial blood supply to the point of gangrene gives the most fulminating picture in obstruction; that loss of salts is often an important factor but one which is easily combated; and that dis-

One should always remember that in folded loop obstructions there is not only obstruction to the main continuity of the gut, but also an additional obstruction of a loop. The latter may become distended quite rapidly, endangering either the terminal or the primary blood supply of the loop, and causing a fulminating picture.

Again, there is the possibility of fairly early rupture or increased permeability of the large bowel, due to distention in the large gut obstructions, allowing fatal peritonitis. An understanding of the mechanics of these different types allows for fuller

appreciations of the potential variability of signs and symptoms.

Early Signs and Symptoms. Our most reliable finding in direct lumen obstructions is available through the use of the *x*-ray. It has been repeatedly demonstrated that gas and fluid levels in the small bowel become characteristic in these direct lumen obstructions in three to six hours. However, we must appreciate that this finding may not be present in some of the conditions where the lumen is not directly closed, as in some venous mesenteric and arterial mesenteric occlusions. Also we know that fluid levels will occur in indirect lumen obstructions as in paralytic ileus. Actually we cannot list unqualifiedly any of the signs and symptoms as being pathognomonic for all types of conditions listed under the intestinal obstruction heading. But *with limitations as previously discussed,* we may list early signs of obstruction as being vomiting, pain, tenderness to deep pressure, obstipation, and fluid or gas levels to roentgenologic study. Increased peristalsis is also usually present at an early stage in the direct lumen obstructions. These signs and *these only* should be considered as at all reliable early ones.

Under *late signs and symptoms* may be included distention, decreased peristalsis, marked rigidity (peritonitis), evidence of alkalosis or acidosis, and collapse, with some of the early signs and symptoms, of course, being carried over into the later picture.

CONCLUSIONS

1. As far as is possible the differences in the signs and symptoms as they appear in the fundamentally different types of intestinal obstruction are identified and discussed in this analysis of 200 cases. An effort is made to determine the time of appearance and consistency of signs and symptoms in the different types and to correlate these findings with mortality rates. The unnecessarily high mortality of obstructions is in part due to failure to make a more clear-cut distinction between early and late signs and symptoms; and in part to failure to appreciate the potential variability of the pathologic physiology of different conditions ordinarily listed under the intestinal obstruction heading. The potential scarcity of clear-cut objective clinical findings in early stages should be more fully realized. Too frequently we wait for clinically pathognomonic signs and symptoms to develop without appreciating that such signs and symptoms may appear only late in the pathologic process when a high mortality with any type of present day treatment is inevitable.

2. It is shown in this series that distention, a sign so frequently given as of early occurrence, often does not appear clinically

TABLE V

DISTENTION

(As to whether reasonably certain clinically absent or present in particular twelve-hour periods)

	Simple Obstructions		Folded Loop Obstructions		Strangulated Hernias	
	Reasonably Certain Present	Reasonably Certain Not Present	Reasonably Certain Present	Reasonably Certain Not Present	Reasonably Certain Present	Reasonably Certain Not Present
First 12 hours	0	7	Possibly 2	5	0	10
Second 12 hours	0	4	4	5	1	5
Third 12 hours	1	4	3	3	1	3
Fourth 12 hours	2	2	4	Possibly 1	Possibly 2	1
Fifth 12 hours	3	0				

until sufficient time has passed to presage a much higher mortality than would have obtained had treatment been given before the distention was clinically demonstrable.

3. The seriousness of folded loop obstructions is illustrated by the mortality figure (71.4 per cent) which is approximately 12 per cent higher than that for simple obstructions (59 per cent). This is true even though the folded loop obstructions were operated on about twenty hours (average time from onset 37.45 hours) earlier than the simple ones (average time from onset 59.5 hours).

4. The gravity of gangrene is indicated by a mortality in this series of 100 per cent.

REFERENCES

1. Donaldson, J. K. Intestinal obstruction; an attempt at an improved discussion of its pathologic physiology and treatment based upon a simplified classification and on past experimental and clinical experience. *Arch. Surg.*, 35: 155 (July) 1937.
2. Donaldson, J. K., and Stout, B. F. Mesenteric thrombosis: arterial and venous types as separate clinical entities. *Am. J. Surg.*, 29: 208 (Aug.) 1935.
3. Knight, G. C., and Slome, D. Intestinal strangulation. *Brit. J. Surg.*, 23: 821, 1936.
4. McIver, M. A. Acute intestinal obstruction. *Am. J. Surg.*, 19: 164 (Jan.) 1933.

The length of life in obstructive jaundice is always limited. . . . Bile loss seems to be far afield from the toxicity of bile. The toxicity is due to the absence of bile, not to its presence.
From—"Bile—Its Toxicity and Relation to Disease" by O. H. Horrall (University of Chicago).

ACUTE ADRENAL CORTEX EXHAUSTION AND ITS RELATIONSHIP TO SHOCK

FRED ROMER REED, M.D.

Associate Attending Obstetrician, Highland Park General Hospital

DETROIT, MICHIGAN

THE question of the adrenal cortex as an important factor in the vast endocrinologic hook-up has been suggested, but whether due to lack of pure potent extract or to a lack of knowledge of its functions, it has been more or less regarded as insignificant by investigators, as well as by average practitioners. One of the first to recognize its importance was the late Dr. A. Scott Warthin, formerly professor of pathology at the University of Michigan, who, in autopsy reports of patients dying from shock, never failed to mention the condition of the adrenal glands and the state of exhaustion present therein. Wolf[1] has suggested the subtle but vital importance of the adrenals a number of times in his comprehensive discussions of the other glands of the endocrine chain. Various writers and pathologists have, from time to time, mentioned possible functions and effects of the adrenal cortex, but through diligent searching, it is found that little accurate information regarding the functions and secretions can be obtained.

In considering a new subject of this type, it is deemed appropriate to review briefly the sequences of thought and experience which have developed and rationalized the idea, and have led to the use of adrenal cortex extract as a treatment for the prevention and relief of the age-old condition, surgical shock.

During the past few years, much progress has been made in the study of the endocrine glands and their effect on the human body in health and disease. We know that in perfect health, all of the endocrine glands are in a correlated balance, which in turn gives a healthy, normally functioning body and mind. We know also that there are many different conditions of modern life which affect the human body and produce either increased function or exhaustion of one or more of these interrelated glands with grave and serious effects upon the body as a whole.

Of these, probably the best understood interrelationships are the gonad-pituitary complex and the hyperthyroid complexes, with their far-reaching physical and mental effects. The pancreatic complex and insulin-secreting disturbance, commonly known as diabetes and various other conditions such as gigantism, acromegaly, dwarfism, etc., are well established syndromes in the medical world. At the present time it is a conceded fact that the endocrine glands, each having a definite function of its own, are so closely interrelated in their actions upon the body that they function in conjunction with each other and are interdependent, often requiring activating or complementary secretions or hormones for full function.

Much research has been done on the function of the adrenal gland and its anatomic parts, the medulla and the cortex. Adrenalin, the secretion of the medulla, has been known and used extensively for many years. On the other hand, the cortical portion and its functions remained a mystery, with the exception of one fact: medical men knew that life in its higher forms could not exist for more than a limited number of hours when all of the cortical tissue had been removed or destroyed.[2]

Addison's disease has been known to be due to a progressive destruction of the adrenal gland, generally as a result of tuberculosis. It was a hopeless condition, controllable only since the recent per-

fection of a satisfactory adrenal cortex extract,[3] the lack of secretions of the medullary portion of the gland being

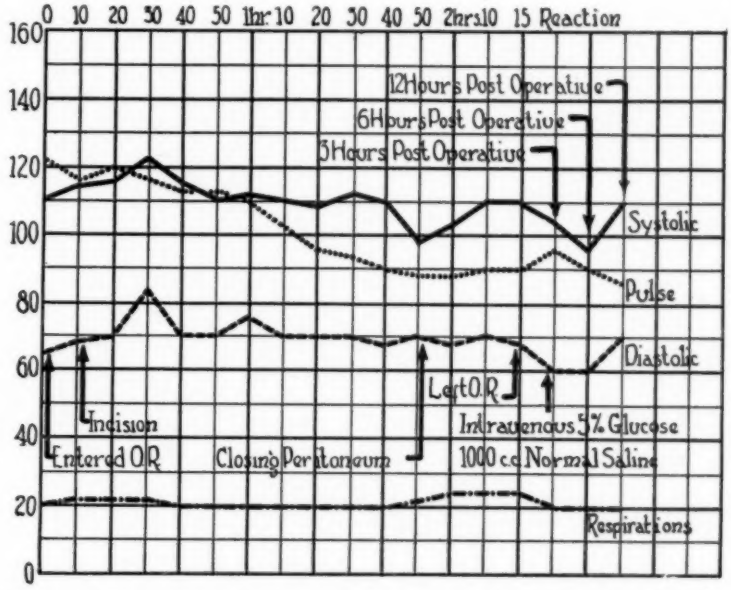


Fig. 1. Case 1. Operative period. Closure of colostomy. Fecal fistula following postoperative gas gangrene (one year previous). Extensive destruction of muscle present. Colostomy into sigmoid. General condition on admission fair. Nitrous oxide and ether anesthesia.

compensated for by the secretions of the thyroid gland.[4] Studies of Addison's disease and research on adrenalectomized animals have shown definite blood chemistry changes similar in both instances, as well as other related clinical findings. The chemical findings most marked are a lowering of sodium and the blood chlorides,[2,5,6,7,8,9,10] an increase in non-protein nitrogen, a decrease of the plasma in the blood stream,[11] and a disproportion from the normal increasing as death approaches. The clinical picture is one of asthenia, beginning with progressive weakness, followed by falling blood pressure, skin pigmentation, edema, failing kidney function, unequal distribution of the blood volume in the vascular system, anorexia, nausea and vomiting, rapidly failing heart action, embarrassed respirations, great physical weakness, coma and death.

Acute conditions, clinically similar to Addison's disease (with the exception of the pigmentation), are frequently seen in their terminal stages; (2) surgical and traumatic shock; and (3) combinations of both causes. I cite the first cause only to mention it and its possibilities in the way of treatment. My own clinical experience has been limited in this field. It is with the second and third types of cases that I have become familiar, those of surgical shock with or without complicating infections of a surgical type.

Much time and study has been given to the subject of shock, in its varying degrees, throughout the history of medicine and surgery. Many volumes have been written, many new theories evolved, and many new treatments used in an effort to overcome this one great cause of mortality. Causes of shock may be classed in four main groups: (1) traumatic, (2) surgical, (3) toxic, and (4) psychic. These causes, singly or collectively, are capable of producing reactions in either humans or animals of a severity sufficient to produce death.

It is an accepted fact that death from shock, whatever its direct cause may be, produces a well defined syndrome, namely: loss of blood volume, either from hemorrhage or a rapidly progressing abnormal distribution of the blood volume within the circulatory system. This is due to loss of vascular tone, with diffusion of the plasma through the capillary walls, and stagnation of the cellular elements in the small capillaries. This in turn produces a rapidly falling blood pressure with decreased nutrition and oxygenation of all body tissues, embarrassment of heart action due to lack of blood volume with which the heart muscle works, and finally death from inanition of the vital cells of the body.

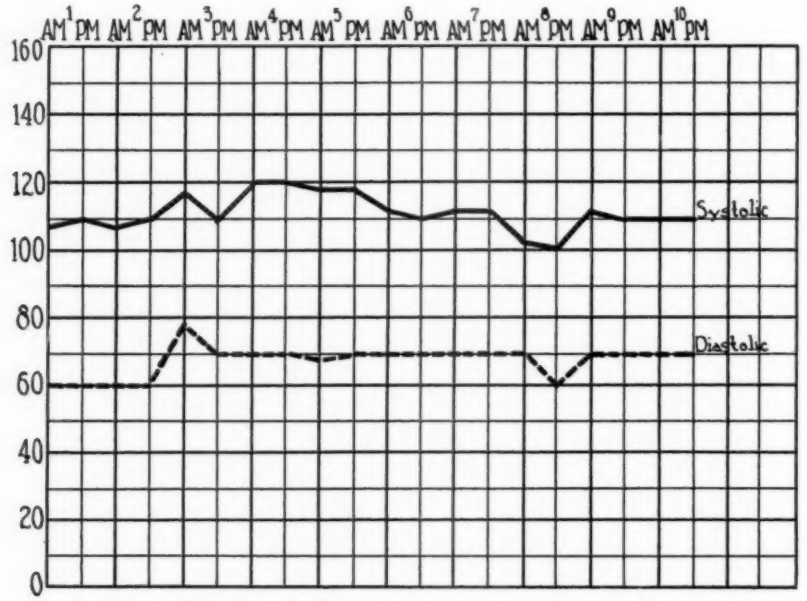


Fig. 2. Same patient as in Figure 1. Postoperative period. Illustrates "settling" of blood pressure to normal following surgery.

Numerous authors have advanced plausible theories as to where this vascular dilatation takes place in the body. The earlier school of thought stated that there was a dilatation of the vascular system in the splanchnic area with pooling of blood at that point. This has been gradually abandoned as pathologists failed to find any great distention of these vessels at autopsy in patients who had died from shock. During and after the war, Quenu[12] and other French and English observers devoted much thought and research to toxic absorption from traumatized areas, and its effects in the production of delayed traumatic or surgical shock.

From their observations, there can be no doubt that the absorption of toxins from degenerating necrotic traumatized areas is a factor in shock production. However, the research of Cannon,[13] Simonart[14] and others has shown that this factor alone is not sufficient in itself to produce the syndrome in its entirety, except in very extensive tissue destruction. Even then, the elements of pain and other psychic reactions are present and contribute to the total syndrome picture.

In 1933, Norman Freeman[15] called attention to the pain factor in the production of shock, and with his experiments proved that when the nerves from the traumatized areas were severed, the shock was greatly reduced. He further showed that the sympathetico-adrenal system was quickly affected in shock, causing vasoconstriction of all vessels in the body except those of the heart and skeletal muscles.

In 1925, Cannon and Britton[16] demonstrated the fact that animals manifesting sham rage secreted an abnormal amount of adrenalin into their own blood streams and developed the clinical shock syndrome,

reducing the amount of blood in the current circulation with falling blood pressure, and at the same time producing constric-

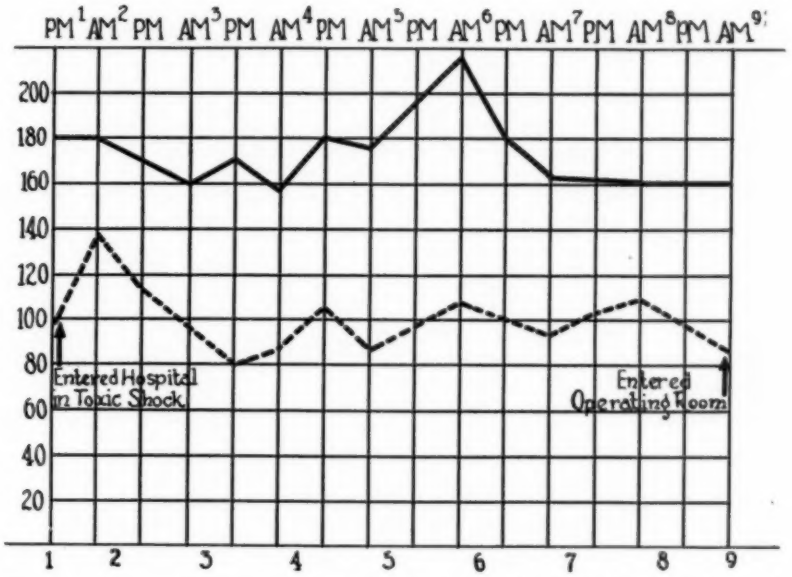


Fig. 3. Case III. Preoperative. Acute cholecystitis, lithiasis, chronic toxic myocarditis, and acute nephritis. Cholecystectomy performed under nitrous oxide and ether anesthesia.

tion of the cutaneous and splanchnic blood vessels, with dilatation of those in the skeletal muscles. Krogh[17] further showed an enormous opening of the capillary beds in these areas with outpouring of the plasma through the capillary walls into the spaces between the muscle fibers, and stagnation of the cellular elements in the capillaries in these muscle areas. Freeman further demonstrated that the continuous injection of adrenalin into animals over a period of two hours lowered the blood volume as much as 27 per cent and produced the shock syndrome.

Prior to the past five years, very little was known in regard to the part which might be played in the shock syndrome by the cortical portion of the adrenal gland. As before mentioned, references had been made to the possibility of this gland being involved in the shock syndrome for a considerable number of years by Dr. Warthin in his autopsy reports. More recently, Crile[18] and his associates have shown that in patients who died of shock, microscopic section at autopsy showed a definite disintegration of the nuclei with granulosis within the cell membranes of three types of body cells, the Purkinje cells of the brain, the interlobular cells of the liver, and the cortical cells of the adrenal glands. Other research reported during the last few years has revealed definite changes in the blood picture in adrenal cortical insufficiency, namely, lowering of blood chlorides and sodium with increase of non-protein nitrogen and dehydration, which again correlates with the blood changes noted in surgical shock.

In 1933, Loeb and his associates,[5,7] as well as Harrop and his associates,[6,10] reported that in adrenal insufficiency, there was a loss of sodium content in the blood stream, accompanied by dehydration. Stewart and Rogoff,[9] and Harrop also reported that the administration of fluids and sodium salts to adrenalectomized animals was of distinct benefit both in correcting the clinical condition and in prolonging the life of the animals. They further showed that with the administration of cortical extracts they were able to obviate these profound water and salt balance disturbances. The recent researches of Maddock and Coller on water balance and

dehydration in surgery[19] have definitely shown that surgical shock is reduced and controlled to a high degree by the properly

From a careful search of the literature thus far, reports of the application of the cortical hormone in the treatment of

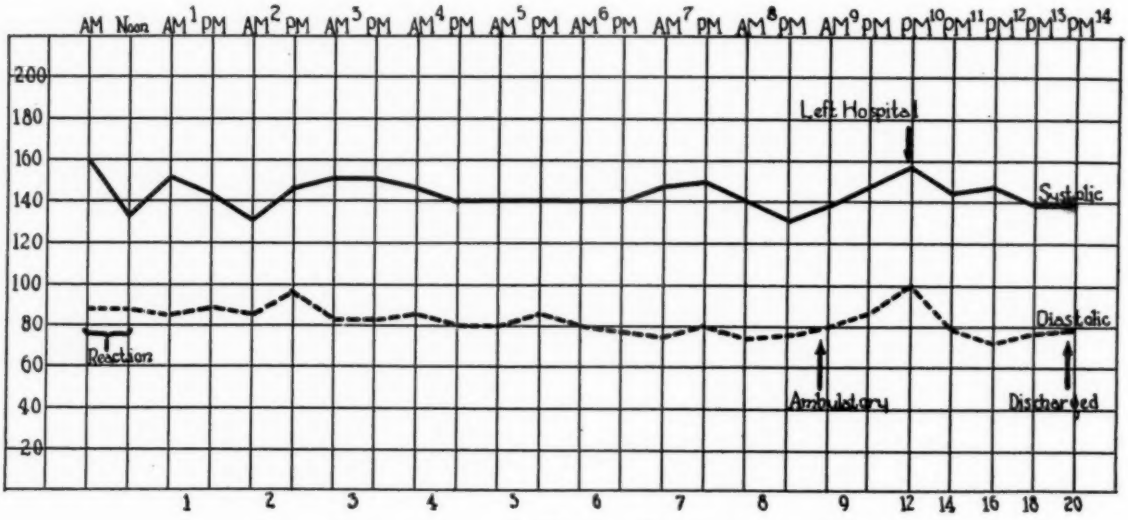


Fig. 4. Same patient as in Figure 3. Postoperative period.

maintained balance of fluid, blood chlorides, sodium, potassium and calcium, which again suggests a possible relationship existing between the known adrenal cortex deficiencies in animals and surgical shock.

Many investigators have maintained adrenalectomized dogs in apparently normal physiologic condition for long periods of time by the daily administration of sufficient amounts of the extract of the adrenal Cortex.[20,21] These authorities have pointed out that the syndromes of anaphylactic shock, like those of adrenal insufficiency, can be alleviated or postponed by the administration of sodium salts, and prevented by the administration of the cortical hormone.

In surgical and traumatic shock the syndromes and blood chemistry findings are very similar to those encountered in adrenal cortical insufficiency in animals. Swingle and his associates[22] have given a detailed comparison of the two conditions, and have reported experiments on dogs which would indicate that the hormone might be of value in the treatment of surgical shock.

shocked conditions have been very limited, while there are a few reports on the use of cortical extract in the treatment of severe infections, toxemias and allied toxic shock. Zwemer,[23] in 1931, reported the use of adrenal cortex extract in the treatment of severe cases of intestinal intoxication in babies, followed by marked improvement within twenty-four hours. Bamberger and Wendt[24] in 1935, reported a beneficial action of the cortical hormone in the treatment of the circulatory system, which had been weakened by diphtheria. Wilson and associates,[25] in 1936, reported the use of the adrenal cortex extract with very encouraging results in the treatment of the acute toxemia and shock following extensive burns. Wenner and Cone[26] reported the improvement of severe pyogenic infections following the administration of from 2 to 7 c.c. of adrenal cortical extract intravenously at intervals of two or more days.

In 1932, I began using desiccated adrenal substances in the treatment of non-surgical nervous, asthenic patients, whose symptoms closely simulated hyperthyroidism, but whose basal metabolic rates were normal or subnormal. In general, these

patients were well nourished individuals whose chief complaints were rapid heart action from the least excitement or moderate exercise and ready fatigue. When subjected to any unusual psychic stimulus, they developed tachycardia, with a profuse cold, clammy perspiration, particularly noticeable in the palms of the hands and the soles of the feet. A coarse tremor of the hands was noted in most instances, accompanied by general muscular weakness. Electrocardiograms showed low voltage curves with either bradycardia or normal heart rates.

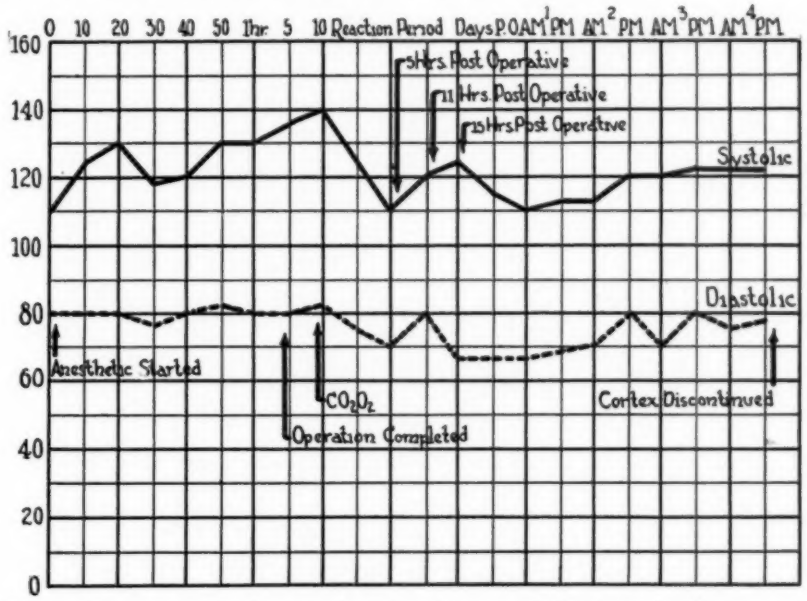


Fig. 5. Case IV. Appendectomy for chronic appendicitis in male of 23 years. Nitrous oxide and ether anesthesia.

It was first thought that these patients belonged in the class of thyroid exhaustion, and small doses of desiccated thyroid substance were prescribed. This treatment aggravated the condition, however, and was therefore discontinued.

A diagnosis of hypo-adrenia was then made and the patients were given desiccated whole adrenal substance. This produced aggravation of a type which indicated that these patients were receiving an excess of adrenalin. Desiccated cortical substance was substituted with favorable results. Their symptoms were gradually alleviated, with a slow rise in blood pressure to normal. Mental tranquillity was restored and later electrocardiograms showed normal heart action in all instances.

The striking similarity between the symptoms exhibited by these patients and those of a patient going into gradual surgical shock led me to believe that desiccated adrenal cortex would be of benefit in preparing asthenic patients for surgery. A series of such cases was started and I noted less shock from operation than was present in referred patients not prepared in this manner.

From the similarity of clinical pictures exhibited in shock and in known cases of adrenal cortical insufficiency (Addison's disease); from the comparison of the blood chemistry findings in adrenalectomized animals and patients in surgical shock; as well as from my own clinical experience in the treatment of hypo-adrenia patients and their preparation for surgery with pure desiccated adrenal cortex substance, I decided to use adrenal cortex extract in the first case of extreme surgical shock presenting itself.

In January 1936, a pelvic surgical patient (Case I) developed a fecal fistula accompanied by an extremely virulent infection in the abdominal incision. She

rapidly became toxic and went into shock. The infection was spreading in spite of all known methods of treatment. I decided

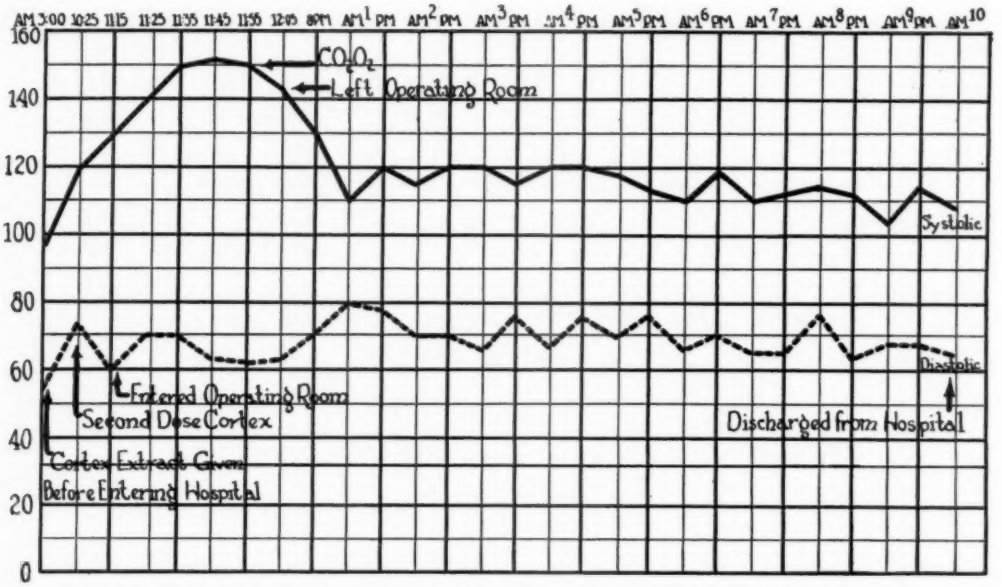


Fig. 6. Case v. Ruptured ectopic pregnancy. Entered hospital in shock. Approximately 500 c.c. of free blood in abdomen. Nitrous oxide and ether anesthesia.

to draw blood for chemical determination of the chloride and non-protein nitrogen contents, and at 5 P.M., the laboratory findings were: blood chlorides 340 mg. per 100 c.c. of plasma; non-protein nitrogen 59 mg. per 100 c.c. of blood. At 6 P.M., a large dose of adrenal cortex extract was administered. At that time, the patient was comatose, with all the typical signs of deep shock, and the relatives were notified that she would not live through the night. At 11 P.M., she was conscious and coherent. Blood chemistry at that time revealed a striking difference from the 5 P.M. samples. The blood chlorides were 528 mg. per 100 c.c. of plasma, and the non-protein nitrogen 30 mg. per 100 c.c. of blood. It should be mentioned that no treatment or medication other than the injection of adrenal cortex extract was administered in the interval between 5 P.M. and 11 P.M. Twenty-four hours later, typical clinical signs of gas gangrene infection became apparent. Blebs developed under the skin and the crepitation extended well out from the incision to the right. Cultures proved positive for B. welchii. Gas gangrene antitoxin was given frequently in 5,000 and 10,000 unit doses intravenously. Four thousand c.c. of fluids were administered daily and adrenal cortex extract, 4 to 6 c.c., was given intramuscularly each twenty-four hours. Following the original blood chemistry, several subsequent determinations were made with normal blood chlorides and non-protein nitrogen readings each time. This patient was able to resume her normal employment four months later, and on November 15, 1936, returned to the hospital for closure of a colostomy following the original operation.

This case served as a background for further clinical research, and in March 1936, another case came under my care which added further to my clinical knowledge of the possibilities of this type of treatment. A young boy (Case 11) entered the hospital with a diagnosis of acute appendicitis, having been referred by another physician. He was acutely ill and was operated on at once. The appendix was found to be ruptured and general peritonitis developed. Again adrenal cortex extract was used postoperatively. The patient responded and recovered rapidly.

Analytic studies of these two cases convinced me that this potent substance should be of value in fortifying all surgical patients for the impending shock. I decided to use intramuscular injections of adrenal cortex extract postoperatively, and later, both before and after surgery, to see if the ordinary surgical shock could be avoided or at least lessened.

Studies were made of blood chemistry and blood pressure, as well as the clinical aspects of the cases postoperatively, with striking results:

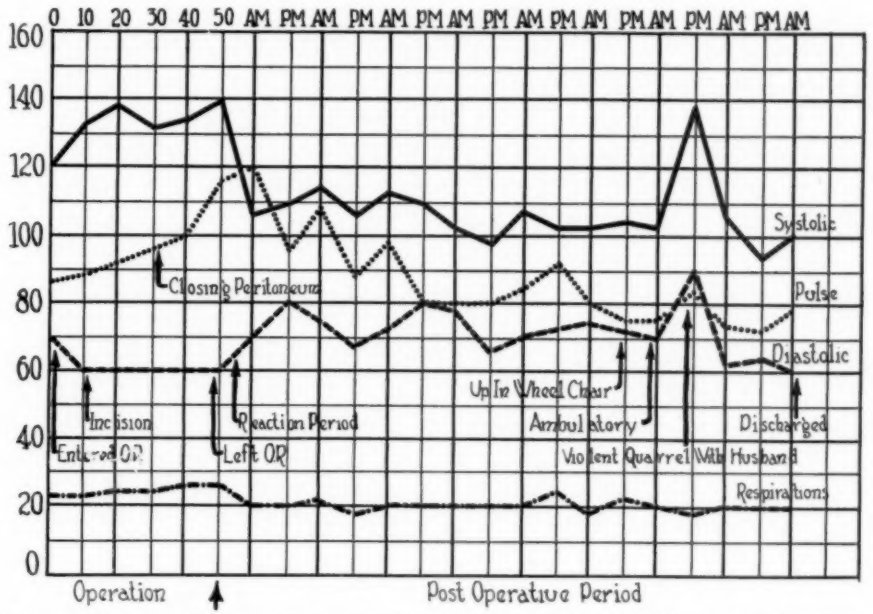


Fig. 7. Case VI. Gangrenous appendix with localized peritonitis. Appendectomy performed and drains inserted. Nitrous oxide and ether anesthesia. Condition of patient on admission poor. Appendicitis occurred three weeks post-partum.

From clinical observations on these early cases, a technique has been developed in the use of adrenal cortex extract in the prevention of surgical shock. Intramuscular injection of from 1 to 2 c.c. is given at an interval of from eight to eleven hours preoperatively, and a second injection of 1 to 2 c.c. one and one-half hours prior to operation. All drugs of a sedative nature which of themselves would produce a drop in blood pressure have been eliminated from the preoperative preparation. Adrenal cortex extract is given postoperatively in 1 c.c. doses at intervals of from six to twelve hours during the early postoperative period. The time of discontinuance of this treatment depends entirely upon the patient's condition and the blood pressure readings.

1. Blood chlorides and sodium were maintained at or near the same point in all cases where the chemistry could be obtained, thus giving a normal balance of body electrolytes and undoubtedly maintaining normal tissue osmotic reactions. This chemical factor is again of great value in the maintenance of normal postoperative water balance both in tissues and in the blood stream.[19]

2. Blood non-protein nitrogen was maintained at or below the preoperative levels.

3. Blood calcium and phosphorous were run in a few cases and showed no appreciable change.

4. Heart action has been stabilized and fibrillation eliminated throughout the time that the cortical extract was administered. (Cases III and VIII.)

5. In one case, it has been noted that by the injection of 5 c.c. of adrenal cortex extract, a urine with a three plus albumin reaction became free of albumin and casts in eighteen hours. One thousand c.c. of

5 per cent glucose in normal saline was given intravenously in conjunction with the adrenal cortex extract during this levels were maintained at or near the preoperative level throughout the operation and in the postoperative period. The usual

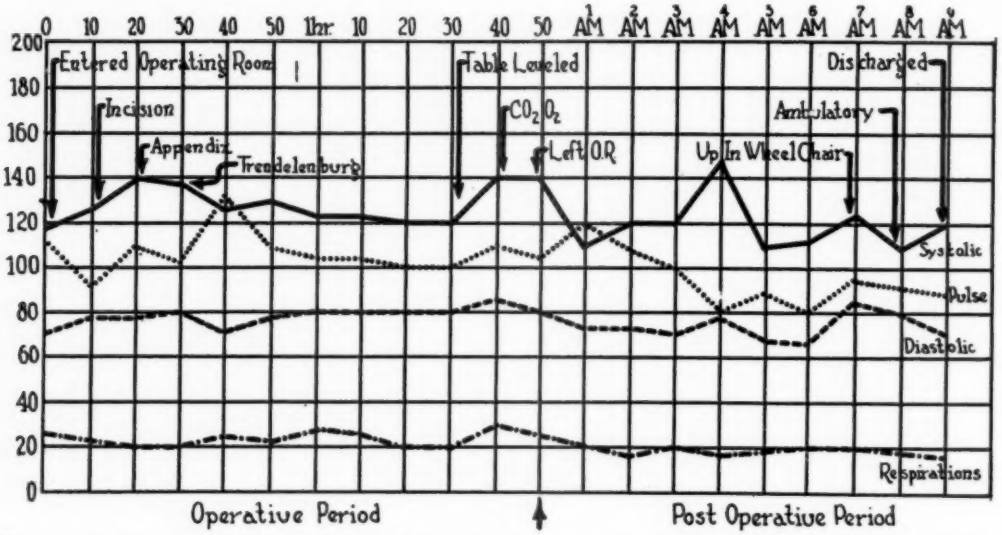


Fig. 8. Case VII. Pyosalpingitis, multiple infected ovarian cysts, pelvic adhesions, chronic appendicitis. Bilateral salpingectomy, left oophorectomy and appendectomy performed under nitrous oxide and ether anesthesia.

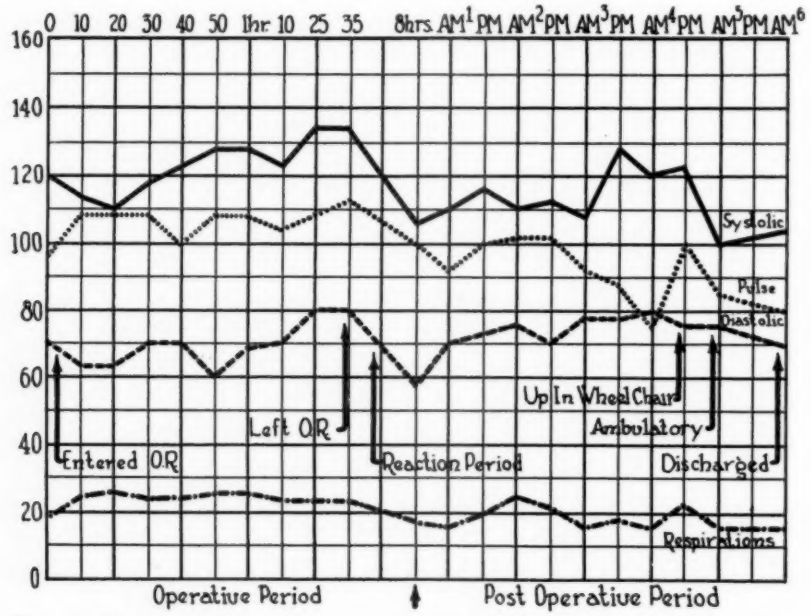


Fig. 9. Case VIII. Thyroid adenoma. Subtotal thyroidectomy done under nitrous oxide anesthesia.

period, but no other medication was used.

In patients who were given adrenal cortex extract in large doses at intervals of eleven hours and one and one-half hours preoperatively along with the usual preoperative preparation, blood pressure curve during this operating period shows a proportionate rise in both systolic and diastolic pressures, with the maintenance of a normal pulse pressure throughout the operative period, followed by a slight drop during the postoperative reaction period and a restoration to the normal preopera-

tive level within a very few hours. (See graphs.) Pulse rates showed little or no acceleration from the preoperative readings during both the operation and postoperative reaction from anesthesia. Loss of blood fluid by excessive diaphoresis has not been noted in any of these cases, and the usual postoperative period of sweating during the reaction from the anesthetic has been avoided in all cases. Dehydration has not been noted clinically in any of the cases. Abdominal distention, gas pains, and other signs of a decreased peristalsis of the abdominal viscera have not been present. Paralytic ileus has not been seen in any of the cases. Fluids and solid foods have been retained early and comfortably, with a quick return of the normal appetite in all cases.

Healing of the incision has been rapid, with the early formation of firm, narrow scars noted in all cases. Scars which have been examined from four to six months later have shown no spreading or widening of the scar area, and maintain a narrow, normal texture. The general appearance of these patients has been good at all times. The skin has been normal in color, even when reacting from the anesthetic. The patients have been mentally relaxed, calm and coöperative at all times. Arterial and venous circulation has appeared normal throughout the body, with no areas of edema showing at any time.

There has been a notable decrease in the pain experienced by these patients during the postoperative period, and considerably less narcotics have been needed to control this factor than I formerly found necessary in similar types of cases. Resistance to infection has apparently been increased, as evidenced by Cases I, II, and III, and it is also to be noted that in Case I, there was never any anaphylactic or serum reaction noted, in spite of the fact that frequent injections of gas gangrene antitoxin were given. In one case it was observed that there was a marked relief from hypertension where this treatment was used after the removal of an infected gall-bladder with cholelithiasis. (Case III.)

The cortical extract used in this series of cases was prepared according to the method of Cartland and Kuizenga.[27]

The case studies with blood pressure graphs accompanying this article have been taken from actual clinical work comprising fifty-two cases seen over a period of fifteen months.

CASE REPORTS

Case I. Female, age 43. Admitting diagnosis, chronic pyosalpingitis.

History. Chronic pelvic inflammatory disease had been present for several years. Colpotomy had been done three times during the past six years in the home. Free drainage of pus was obtained on the first two occasions, but none was obtained at the last operation, in October 1935. The patient was referred to me on December 27, 1935. There was no preoperative administration of adrenal cortex extract.

Operation. Bilateral salpingo-oöphorectomy and routine appendectomy were done. On the second postoperative day, gas and fecal matter were expelled per rectum. Following this, the abdomen became distended, and there were nausea, vomiting and hiccough. Profuse drainage of pus from the incision began on the third day. The nausea and vomiting persisted and a Levine tube was inserted. Intravenous glucose and saline were given freely throughout the second and third days. On the fourth day, there was a distinct fecal odor to the drainage, but no fecal particles were present. On the sixth day, the patient became comatose and went into severe toxic shock.

Blood chemistry at 5 P.M. revealed: blood chlorides at 340 mg. per 100 c.c. of plasma; non-protein nitrogen 59 mg. per 100 c.c. of blood. Adrenal cortex extract, 2 c.c., was given intramuscularly. At 11 P.M., the patient was conscious and coherent. Blood chemistry determinations showed blood chlorides 528 mg. per 100 c.c. of plasma; non-protein nitrogen 30 mg. per 100 c.c. of blood. The patient did not receive any other medication or fluid either by mouth or intravenously during the period from 5 P.M. until 11 P.M.

Definite clinical signs of gas gangrene infection developed in and around the incision on the eighth day and cultures were taken. Gas gangrene antitoxin was given intravenously in 5,000 and 10,000 unit doses at twelve-hour intervals, and the body fluid intake was measured to meet 4,000 c.c. daily. The amount which could not be taken by mouth were given as 5 per cent glucose in normal saline intravenously. Adrenal cortex extract was given at regular intervals, an average of 4 c.c. daily being maintained.

The temperature ranged from a high of 104.6 degrees to a low of 101 throughout the five days following the development of gas gangrene, and then gradually subsided. The areas of gas gangrene blebs and the subcutaneous crepitation reached a maximum on the third day following the recognition of infection. At that time, crepitation extended upward to the right axilla, well back over the dorsal surface of the body, nearly to the midline and down over the crest of the right ilium. The temperature gradually subsided and reached normal on the eighteenth postoperative day, where it remained, except for occasional subnormal periods.

The convalescence was gradual; the patient recovered and left the hospital seventy-one days following her admittance.

She returned to normal work about four months following the original operation. On November 15, 1936, she returned to the hospital for closure of the colostomy resulting from the first operation. She was prepared for three days and on November 18, 1936, the old scar was removed from the abdominal wall, masses of adhesions, including the omentum and six loops of the ileum were dissected free from the scar area. The colostomy was dissected free from the scar in the abdominal wall and closed. The abdominal wall was then closed, repairing the ventral hernia resulting from the gas gangrene infection, using fascia to replace the right rectus muscle which had been completely destroyed by the infection. Complete blood pressure records of this operation are presented. (Fig. 2.) During the second operation, this patient did not show shock at any time. Her recovery was uneventful and on the eighteenth post-operative day, she returned by train to her home, 150 miles distant.

Laboratory reports of the first operation showed cultures from the wound positive for B. welchii, using Wilson and Blair medium.

Case II. M. R., male, age 9 years, was referred by another physician. The diagnosis on admission was acute appendicitis, ruptured appendix with acute peritonitis.

No preoperative preparation with adrenal cortex extract was given. The patient was given a routine abdominal preparation and operated on immediately upon entrance to hospital. The ruptured abscessed appendix was removed. Generalized peritonitis with free pus in the abdomen was present, and there was no protective walling off of the area by omentum. Multiple drains were placed in the abdomen and the usual closure made.

Postoperative Period. The abdomen was distended and tense at the end of twenty-four hours, with nothing passing by rectum. After thirty-six hours the abdomen was still distended and hard, with nothing passing by rectum, despite the fact that hot stoops had been applied continuously; warm enemas, pitressin, and rectal tubes were used without results. At that time adrenal cortex extract was given in 1 c.c. doses every six hours and the other treatment continued. Twelve hours following the administration of adrenal cortex extract the bowel was opened and the abdomen soft. Free drainage of pus was established and the general condition of the patient improved.

During the postoperative period to this point, the patient had received by intravenous injection an average of 2,000 c.c. of 5 per cent glucose in normal saline each twenty-four hours. However, following this period, the patient was coöperative and fluids were freely taken by mouth. The recovery was prompt and uneventful.

Case III. A. A., female, age 56 years. The diagnosis on admission was infective cholecystitis with cholelithiasis.

The present illness had begun six days previous. On entrance to the hospital the patient was in shock and comatose. Her blood pressure was 180/98; the heart showed fibrillation and irregular extrasystoles. The abdomen was distended and rigid. Laboratory findings on admission showed the following:

Urinalysis: reaction acid; color amber; specific gravity 1.028; albumin three plus; sugar negative; few pus cells and numerous finely granulated casts present.

Blood Count: hemoglobin 90 per cent; red blood count 4,950,000; white blood count 19,000; differential count filament cells 60 per cent, non-filament cells 15 per cent, lympho-

cytes 20 per cent, endothelial cells 3 per cent, eosinophile cells 3 per cent.

Blood Chemistry: Non-protein nitrogen 34 mg. per cent; blood chlorides 578 mg. (NaCl) per cent; icterus index 3.3. Kahn test negative.

Treatment. Directly following the drawing of blood for laboratory determinations, the patient received 1,000 c.c. of 5 per cent glucose in normal saline solution intravenously, and 3 c.c. adrenal cortex extract intramuscularly. Two hours later the patient was taking fluids freely by mouth and retaining them. The bowel was opened with warm enemas, and five hours following the first injection of adrenal cortex extract, when a second injection of 2 c.c. was given, the patient was resting quietly and comfortably. During the night fluids were forced by mouth whenever the patient was awake, and the following morning she was conscious and coöperative. A second urinalysis at this time gave results as follows: reaction acid; color amber; albumin negative; sugar one plus positive; few pus cells present. No casts in specimen.

The abdomen at this time was soft and relaxed except for tenderness and moderate spasm of the muscle in the upper right quadrant. The heart was regular in its action and showed no signs of fibrillation or extrasystoles. The third day in hospital, the general condition was improving, with the patient taking soft food and fluids freely without any discomfort. Urinalysis then showed reaction acid; color amber; albumin negative; sugar negative; few pus and epithelial cells present. This would indicate that the sugar present in the second sample was simply a spilling of glucose from the intravenous injection of the first day in the hospital, and not evidence of an abnormal kidney function.

During the next five days the patient's condition continued to improve, except for two short intervals, when she apparently passed more gallstones. After careful preparation she was taken to the operating room on the eighth day after entrance to the hospital and cholecystectomy done.

This patient received a maintenance dose of 1 c.c. of adrenal cortex extract at twelve hour intervals during the preoperative period after the first twelve hours in the hospital. Blood pressure readings were not taken in the operating room and for that reason are not included in the graph in this case. (Fig. 3.)

Postoperative Period. The patient was comfortable and showed no distress. There was no period of diaphoresis during the reaction from anesthesia. Fluids were taken and retained in three hours. The abdomen was soft and flat at all times and the bowel opened on the second day with enemas. Recovery was rapid and the patient was able to be up in a chair on the ninth day postoperatively. She left the hospital on the twelfth day, the incision being completely healed and firm.

Adrenal cortex extract was given in 1 c.c. doses every six hours for four days and every twelve hours thereafter until the patient was discharged from hospital. She remained in the city at the home of her daughter for nine days before returning to her home which is about fifty miles from Detroit. Immediately on her return home she opened her gift shop and has continued her normal routine of life without discomfort since that time. (Fig. 4.)

Case IV. F. M. J., male, age 23 years, was admitted with a diagnosis of chronic appendicitis. He had had several acute attacks during the previous five years.

X-ray examination showed that the appendix could not be visualized with barium mixture. The cecum was immobilized, probably from adhesions.

The patient was prepared in the usual manner, except that 2 c.c. of adrenal cortex extract were administered eleven hours, and again one and one-half hours preoperatively. This patient did not at any time show a blood pressure reading below the preoperative point. Readings were taken preoperatively on entering the operating room and at ten-minute intervals during the operation as well as at regular intervals during the convalescent period. (Fig. 5.)

The patient showed no period of diaphoresis during the reaction from anesthesia, and was taking fluids freely three hours postoperatively. The bowel was opened freely on the second day with enemas, and no abdominal distention was noted at any time. The patient was ambulatory on the fifth postoperative day.

Case V. R. R., female, age 26 years was admitted with a ruptured ectopic pregnancy. She was first seen at her home at 3 A.M. in consultation with Dr. H. N. Calkins. Blood pressure readings at that time were 96/54, and she was in moderate shock from loss of blood into the abdomen.

She was transferred to the hospital by ambulance where she received 2 c.c. of adrenal cortex extract intramuscularly and fluids were forced by mouth. Ice caps were applied to the abdomen and the patient rested until 10:30 A.M. at which time the blood pressure was 120/74. She was prepared and taken to the operating room at 11:15 A.M. On entering the abdomen there was a free escape of dark extravasated blood, estimated to be in excess of one pint. An ectopic pregnancy was found in the outer third of the left tube, and a large cyst involving most of the left ovary. The ectopic had ruptured into the abdomen and also into the left broad ligament, where a large hematoma was present. The left tube and ovary were removed and the abdomen closed.

Blood pressure readings were taken on entering the operating room and at regular intervals throughout the entire operation as well as at regular intervals morning and evening postoperatively.

The postoperative period of convalescence was uneventful. There was no postoperative diaphoresis during the reaction from anesthesia. Fluids were taken and retained in three hours. Adrenal cortex extract was administered intramuscularly in 1 c.c. doses at twelve-hour intervals. The patient was ambulatory on the ninth day and discharged on the eleventh day. (Fig. 6.)

Case VI. M. K., female, age 24 years, entered hospital three weeks post-partum, with acute appendicitis.

Her present illness had begun forty-eight hours previously with pain in the lower right abdomen, followed by nausea twenty-four hours later and vomiting about six hours prior to entrance to hospital. Examination on entrance to the hospital revealed marked tenderness and rigidity over the entire abdomen, but most pronounced over the lower right quadrant. Her general condition was not good, early signs of toxic shock being present.

She rested for a period of four hours. She was given 2 c.c. of adrenal cortex extract on admission and again after the four-hour rest period, following which she was prepared and taken to the operating room. On entering the abdomen there was a free escape of milky white fluid from the peritoneal cavity; the omentum was well down over the lower right quadrant of the abdomen and closely wrapped about the appendix to which it was becoming adherent. The omentum was clamped and resected, care being taken not to disturb its attachment to the badly swollen and gangrenous appendix. The appendix was removed in the usual manner and the abdomen closed after two cigarette drains had been placed in the peritoneal cavity. Blood pressure readings were taken at regular ten-minute intervals in the operating room, beginning with the preoperative pressure and ending with a postoperative reading. The patient showed no signs of shock at any time and left the operating room in good condition.

Postoperative Convalescence. There was no period of diaphoresis during the reaction from anesthesia. The patient was taking and retaining fluids in three hours. The abdomen did not show any distention at any time postoperatively. The bowel was opened with enemas on the second day without difficulty. She was ambulatory on the sixth postoperative day and discharged on the ninth day. All drainage stopped on the sixth day and the incision was completely closed at the time of her discharge from the hospital. Complete blood pressure readings were taken at regular intervals morning and evening during the postoperative convalescent period. (Fig. 7.) Adrenal cortex was administered in doses of 1 c.c. at regular twelve-hour intervals.

Case VII. M. R., female, age 29 years, was admitted with a diagnosis of chronic salpingitis, bilateral (pyosalpingitis) and chronic appendicitis. The patient stated that she had experienced repeated attacks of pelvic pain for the previous six years, since the birth of her last child. Her present illness had begun three weeks before, with chills and fever accompanied by pain and tenderness in the pelvis, and rigidity over the lower abdomen. She was put to bed at home and given expectant treatment by her family physician, being transferred to the hospital one week after the temperature had reached normal. Examination on entrance to the hospital revealed fluctuating masses in both lateral cul-de-sacs, the one on the left being larger than that on the right. Preoperative preparation of 2 c.c. of adrenal cortex extract was given at eleven, and at one and one-half hours before entering the operating room.

Bilateral salpingectomy, left oöphorectomy, and a routine appendectomy were done. Numerous adhesions were resected. Two cigarette drains were placed in pelvis.

Postoperative Convalescence. No period of diaphoresis occurred during the reaction from anesthesia. Fluids were taken and retained in three hours. The bowel was opened with enemas on the second day, and no abdominal distention was noted. The patient was ambulatory on the eighth day and discharged on the tenth day.

Adrenal cortex was given in doses of 1 c.c. at regular intervals of twelve hours during the convalescent period. The incision was healed on discharge, the last drain having been removed on the sixth day. Blood pressure readings were taken at regular intervals during the operation and convalescence. (Fig. 8.)

Case VIII. E. P., female, age 21 years, had a thyroid adenoma. She had first noticed an enlargement of the thyroid at the age of puberty. Tachycardia had been present for the past four years. B.M.R., taken two years before, was plus 22. Medical treatment had given relief for short periods, but the tachycardia returned after treatment was discontinued. B.M.R. on entrance to the hospital was plus 25.

The patient was given preoperative medication with iodine for five days before entering hospital. On the evening of entrance to the hospital, 2 c.c. of adrenal cortex extract were given intramuscularly at 9 P.M. and repeated at 6 A.M., before the patient was taken to the operating room at 8 for a subtotal bilateral thyroidectomy.

Postoperative Convalescence. No period of diaphoresis was seen during reaction from the anesthesia. The patient was ambulatory on the fourth day and discharged on the sixth. Adrenal cortex extract was given in 1 c.c. doses at regular twelve-hour intervals during convalescence. Complete blood pressure records were kept during the operation and convalescence. (Fig. 9.)

SUMMARY

In reviewing this series of over fifty surgical cases treated with adrenal cortex extract, the following reactions have been observed:

Body electrolytes, i.e., chlorides, sodium, etc., are apparently maintained at or near normal balance.

Blood pressures are stabilized during both operative and postoperative periods, with pulse pressures maintained at or above normal levels.

Improvement is evidenced in certain cardiac irregularities; i.e., extrasystoles, chronic myocarditis, and auricular fibrillation.

Excessively rapid pulse rates have not been noted.

During the anesthesia reaction period the skin maintains a normal color, warmth and moisture with a marked absence of the usual heavy diaphoresis (obviously barring the use of heavy "ether blankets").

Deleterious gastrointestinal symptoms, i.e., nausea, vomiting, distention and gas pains, are noticeably mitigated.

The amount of narcotics needed to alleviate pain and distress is diminished.

Healing of incisions is expedited, with narrow, firm scar formation.

Accelerated recovery curtails the usually required time of hospitalization and expedites the early return to normal activity.

The above results are obtained with the use of an adrenal cortex extract which is biologically assayed for consistent strength and freedom from adrenalin.

REFERENCES

1. Wolf, William. Endocrinology in Modern Practice. Phila., 1936. Saunders.
2. Swingle, W. W., Pfiffner, J. J., Vars, H. M., Bott, P. A., and Parkins, W. M. *Science*, 77: 58 (Jan. 13) 1933.
3. Schaffer, Robert L., and Strickroot, F. L. Adrenal cortical insufficiency. *J. Michigan State Med. Soc.*, 35: 516 (Aug.) 1936.
4. Crile, G. Diseases Peculiar to Civilized Man. N. Y., 1934. Macmillan.
5. Loeb, R. F., Atchley, D., Benedict, E. M., and Leland, J. *J. Exper. Med.*, 57: 17, 1933.
6. Harrop, G. A., Soffer, L. J., Ellsworth, R., and Trescher, J. H. *J. Exper. Med.*, 57: 17, 1933.
7. Loeb, R. F., Atchley, D., Gutman, E. B., and Jullson, R. *Proc. Soc. Exper. Biol. & Med.*, 31: 31, 1933.
8. Loeb, R. F. *J. A. M. A.*, 104: 2177, 1935.
9. Stewart and Rogoff. *Am. J. Physiol.*, 84: 649, 1928.
10. Harrop, G. A., Soffer, L. J., Nicholson, W. M., and Strauss, M. *J. Exper. Med.*, 61: 839, 1935.
11. Zwemer, R. L., and Sullivan. *Endocrinology*, 18: 97, 1934.
12. Quenue, E. *Rev. chir.*, 56: 204, 1918.
13. Cannon, W. B. Toxic and nervous factors in traumatic shock. *Ann. Surg.*, 100: 704 (Oct.) 1934.
14. Simonart, A. *Arch. internat. pharm. et therap.*, 37: 269, 1930.
15. Freeman, N. *Am. J. Physiol.*, 103: 185, 1933.

16. Cannon, W. B., and Britton, S. W. *Ergebn. d. Physiol.*, 72: 283, 1925.
17. Krogh, A. The Anatomy and Physiology of the Capillaries. New Haven, 1922. Yale University Press.
18. Crile, George. Phenomena of Life. Norton, 1936. Chapter 1.
19. Maddock, W. G., and Coller, F. A. Water balance in surgery. *J. A. M. A.*, 108: 1, 1937.
20. Swingle, W. W. *Medicine*, 11: 371, 1932.
21. Wolfram, J., and Zwemer, R. L. *J. Exper. Med.*, 61: 9, 1935.
22. Swingle, W. W., Pfiffner, J. J., Vars, H. M., Bott, P. A., and Parkins, W. M. *Science*, 77: 58, 1933.
23. Zwemer, R. L. *Endocrinology*, 15: 382, 1931.
24. Bamberger and Wendt. *Klin Wchnschr.*, 14: 846, 1935.
25. Wilson, W. C., Rowley, C. D., and Gray, N. A. *Lancet*, 1: 1400 (June 20) 1936.
26. Wenner and Cone. *Arch. Otolaryng.*, 20: 178 (Aug.) 1934.
27. Cartland and Kuizenga. *Am. J. Physiol.*, 117: 678 (Dec.) 1936.

Operation on patients with obstructive jaundice offers three avenues of danger, aside from the so-called "accidents" of surgery: hemorrhage, uremia and hepatic insufficiency.
From—"Bile—Its Toxicity and Relation to Disease" by O. H. Horrall (University of Chicago).

THE TREATMENT OF SUPPURATIVE CUTANEOUS WOUNDS AND ULCERATIONS WITH COD LIVER OIL AND ALLANTOIN

HARRY A. SALZMANN, M.D., F.A.C.S. AND LEOPOLD Z. GOLDSTEIN, M.D.

Associate Surgeon, Mount Sinai Hospital

Demonstrator of Obstetrics, Jefferson Medical College Hospital

PHILADELPHIA, PENNSYLVANIA

HISTORICAL

THE treatment of wounds has engaged the attention of mankind since prehistoric time. One of the earliest medical records in existence, the Edwin Smith Surgical Papyrus, contains a discussion of the principles to be followed in the treatment of traumatic wounds. The fact that perfection in the treatment of wounds has not yet been attained has evidently constituted a challenge to investigators, judging from the multiplicity of substances brought forward to speed up repair. Through the centuries, various vogues of wound treatment have had their evanescent sway, but no one method has survived the test of time.

The use of living maggots and insects in the treatment of osteomyelitis, abscesses and non-healing wound infections dates back to antiquity. Egyptian, Hindu and Arabian surgeons were not at all unfamiliar with the invasion by maggots of gangrenous and non-healing wounds although it is hardly likely that the medical men of old recognized the therapeutic value inherent in the maggots.

The relation of flies and maggots to disease processes is mentioned, perhaps for the first time in literature, by Ambroise Paré (1510–1590). Paré found gangrenous wounds inflicted during the Battle of St. Quentin (1557) fetid and full of maggots. He employed a recipe of young larvae boiled in oil of lilies as a dressing for gunshot wounds.

Later, in 1704, Zachmann discussed the origin of maggots in wounds, and Larrey, during the Napoleonic wars, noted the healing effects of living maggots in the widespread suppurative wounds of soldiers. This writer was convinced that these insects accelerated cicatrization by shortening the work of nature and by producing an elimination of the necrotic cells by devouring them. He, furthermore, noted that the larvae consumed putrid material and did not disturb any living tissues.

Malgaigne (1847) mentions the use of maggots, referring to Larrey's observations. From this date, little investigative work was done with this form of therapy, until 1928 when Baer commenced the use of maggots in the treatment of osteomyelitis. However, the beneficial effect of the maggots accidentally contaminating major wounds was repeatedly noted by numerous clinicians. Crile, Martin, and others, in 1917, observed that the wounds inflicted in the World War which healed best were those containing maggots.

The therapeutic value of maggots in osteomyelitis and other chronic suppurations has been exemplified in many excellent clinical reports by Baer, Livingston, Robinson, Hewitt, Asbury and others.

ACTIVE PRINCIPLE OF MAGGOT THERAPY

When this form of therapy was first employed, it was assumed that in addition to the mechanical action of the maggots, and the production of serum from local stimulation of the granulation surfaces, an active principle was produced which was responsible for the healing. Robinson, in 1933, conceived the idea that maggots secrete a definite substance which stimulates growth of vascular granulation, cleanses

wounds and brings about a healthy condition in the ulcerated region.

It was also suggested in 1931 (Goldstein) that an enzyme-like product produced by the maggot bodies acted as a digestant and antiseptic. The employment of an emulsion or paste of ground sterile maggots was suggested by this writer for the treatment of superficial wound infections and chronic discharging wounds and indurative ulcers. Recently, Livingston and Prince have reported excellent results in the treatment of chronic osteomyelitis and other ulcerative conditions with the use of extract of maggots. The substitution of living maggots with an extract of the larvae gave results equally as good as the use of the actual maggots.

Fig. 1. Case 1. Large ulcer following removal of epithelioma a few days after institution of cod liver oil-allantoin treatment.

The extract of the maggots is composed of several components each of which is claimed to be the active therapeutic factor, namely: sulfydryl, calcium, cysteine, glutathione and natural allantoin. The sulfydryl group has been considered the growth stimulating agent by several writers, notably Hammett and Reimann, Kendall and Brunsting and associates. Stewart and others attribute the therapeutic effect to the high calcium content which, in combination with picric acid, forms chemically an active healing agent.

The extraction of allantoin from maggots and their excretions offered direct evidence of a most important active therapeutic agent underlying the success of maggot extract therapy. Allantoin has been employed unknowingly from the earliest times in the treatment of chronic non-healing suppurative wounds and burns in the form of a plant root richly laden with allantoin, known as the common comfrey (symphytum officinale). This plant has been recommended by ancient writers in medicine as a dressing for wounds, sores and ulcers of different types. It has received a traditional reputation among the country

folk, both in England and in Ireland, as a domestic herbaceous medicament for the treatment of ulcerative affections.

In 1912 Macalister[23] employed allantoin in the form of an infusion of the powdered comfrey root in the treatment of chronic indolent ulcers which refused to heal with ordinary remedies, and obtained healing effects with rapid granulation. Dr. Tetherly of the Chemical Department of the University of Liverpool, under the direction of Macalister, was the first to prove that the crystalline body he obtained from the root was allantoin $C_4H_6N_4O_5$. This substance may also be obtained synthetically by the alkaline oxidation of uric acid in the cold. It is also a characteristic component of the fetal allantoic secretion.

William Bramwell[4] likewise succeeded in curing old ulcers which had resisted treatment by other means with the simple extract from the comfrey root applied on lint to saturation. Clinical corroboration of the healing effects of allantoin in ulcerative conditions, however, was not obtained until 1935 when Robinson[31] determined that allantoin was a constituent of the urinary excretions of surgical maggots. This investigator tested this substance on chronic non-healing infected wounds with edematous indolent tissues. After the first few treatments, small areas of shining pink granulation tissue was found growing in the wound, followed later by a general development of healthy granulation tissue. Robinson concluded that the excretion of allantoin into the wound is undoubtedly one of the factors contributing to the remarkable healing effects obtained in maggot therapy.

Allantoin is bland, harmless and stable under ordinary conditions. It is odorless and does not stain. Since it is not antiseptic, it does not directly retard bacterial growth; hence the addition of a chemical antiseptic is generally indicated when it is used in non-sterile preparations. The substance is the principal terminal product of purine metabolism in animals below man and it results from the oxidation of uric acid.

THERAPEUTIC EFFECT OF COD LIVER OIL IN ULCERATION

In 1935, several articles appeared describing the therapeutic value of cod liver oil in various combinations in the treatment of cutaneous ulcers. Borovskaya[3] reported rapid healing in fifty-three cases of ulcerative lupus treated with an ointment containing 33 per cent cod liver oil. The bactericidal value of cod liver oil was studied in both animal and human subjects by Tumanski.[37] This author also observed favorable results from cod liver oil, especially in granulating wounds of the soft tissues. Drigalski[7] after extensive bacteriologic experimentation found that cod liver oil has a definite bacteriostatic, as well as bactericidal, action. Furthermore, he believes that the cod liver oil tends to hinder the passage of toxins from the wound into the adjacent tissues. Lundh,[22] in a series of animal experiments,found that only those ointments which contained a high percentage of cod liver oil tended to shorten the healing time of cutaneous ulcers. He further found that the experimental animals showed an improvement in their general condition, and attributed this betterment to the absorption of cod liver oil from the wound surfaces.

Most writers attribute the healing action of cod liver oil to its vitamin A and D content (Hayashi); others attribute this action to the fatty acids. Seiring,[33] premising his procedure on the observations of Dresel, who in 1927 showed that fatty acids have a bactericidal action on gram positive bacteria, treated a number of patients with certain of the high non-saturated fatty acids and obtained results similar to those obtained with cod liver oil.

Many surgeons[14,16,28] have employed cod liver oil ointment in conjunction with plaster splints in the treatment of compound fractures and in fractures complicated by infected wounds of the overlying soft tissues. The concensus of opinion of these observers is that cod liver oil has an extremely favorable effect

on healing and aids in the removal of necrotic tissue. However, it is difficult to evaluate the action of cod liver oil in these

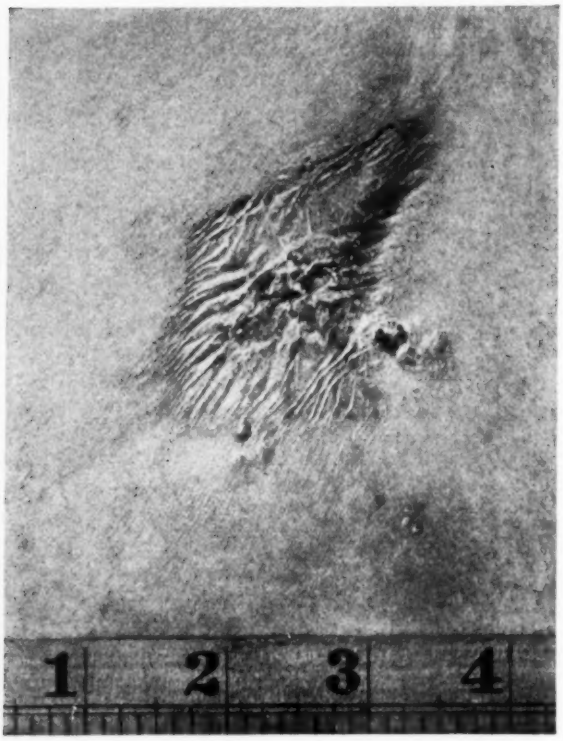


Fig. 2. Case 1. Photograph taken ten weeks after treatment with cod liver oil-allantoin ointment. Complete healing has taken place. A small dark papule immediately external to the scarred area may be evidence of recurrence of the growth.

cases because the results depend on the entire treatment rather than on the cod liver oil alone.

In 1934 Löhr[21] reported that cod liver oil was remarkably effective in controlling secondary infection in burns covering extensive areas. Burns and wounds treated with cod liver oil undergo rapid cleansing and epithelization. Löhr found the cod liver oil superior to tannic acid. He employed the closed method, i.e., the cod liver oil is applied in salve form and the area covered by a plaster of Paris cast. Deeply ulcerated wounds healed rapidly under the influence of the cod liver oil and the enforced rest of the involved part. Steel,[35] impressed by the results of Löhr, used cod liver oil in the treatment of extensive burns. Instead of using the "occlusive" method as described by Löhr, and also by Winnett-Orr, Steel applied the cod liver oil directly to the wound and then covered the latter with lint. The results obtained in burns of varying degree were uniformly excellent. Healing was rapid and attended by a minimum of wound infection.

COD LIVER OIL-ALLANTOIN THERAPY

Since the recognition of the value of maggots as a therapeutic agent in the treatment of suppurative wounds and osteomyelitis, the attention of many workers has been directed to devise a treatment which would embody the active principles of maggots but eliminate the unpleasant features and inconveniences of the maggot treatment. In the present study, we have employed cod liver oil with allantoin and phenol in a number of chronic dermatologic affections, burns, non-healing sinuses following abdominal operations, chronic ulcers, and similar conditions.

The preparation employed in this investigation consisted of an ointment with a lanolin base, containing 45 per cent cod liver oil, 2 per cent allantoin in combination with 0.5 per cent phenol.

The ointment has proved extremely beneficial in chronic, poorly granulating wounds and ulcerations of the skin; in second and third degree burns; in extensive exfoliative types of dermatitis, and in irritative skin conditions associated with pruritus. The process of epithelization is decidedly stimulated, and complete healing is accomplished in a period of time varying from a few days to several weeks in the less severe types of affections, to several months in the widespread deep ulcerative conditions.

In a previous report,[9] it was shown that the addition of allantoin enhances the value of cod liver oil. This was demonstrated in two cases of widespread dermatitis in which the preliminary treatment with soothing ointments containing cod liver oil and phenol did not bring about any improvement. When the combined allan-

toin and cod liver oil preparation was used, noticeable improvement, both objective and subjective, occurred in a few days,

Fig. 3. Case 11. Ulcer of foot in a patient with Buerger's disease before institution of cod liver oil-allantoin treatment.

Fig. 4. Case 11. Appearance of ulcer on December 20, 1937, four months after beginning of cod liver oil-allantoin therapy. The wound is granulating and considerably smaller in size.

and was progressive until complete disappearance of the eruption within seven weeks after institution of treatment.

Applications of the ointment are made at least once a day and the part is covered with sterile gauze. The affected area must be completely covered with the ointment. In those cases where there is danger of too great an absorption of phenol, because of the extent of the lesion, the phenol may be omitted. In less widespread lesions, there is no danger of excessive phenol absorption.

The following are some of the conditions in which an ointment containing cod liver oil (45 per cent), allantoin (2 per cent), and phenol (0.5 per cent) was employed by us: one case of deep, indolent, residual ulcer following removal of an epithelioma of the back by surgical diathermy and electrocautery; three cases of non-healing sinus tracts following abdominal operation; one chronic infected ulcer of the foot associated with Buerger's disease; two cases of ulcer of the foot associated with diabetes; two cases of varicose ulcer; one sinus following incision of an ischiorectal abscess; one case of osteomyelitis of the distal phalanx of finger; three cases of second and third degree burns; three cases of anal pruritus, and three cases of widespread dermatitis.

CASE REPORTS

Following are abstracts of the clinical histories of some of the representative cases treated in our series:

Case 1. S. A. T., white male, age 78, first noticed a small verrucous growth on the mid-dorsal region of the back about seven years ago. The growth continued to increase in size and became ulcerated. He was treated with various salves without improvement. Following a course of intensive *x*-ray irradiation, the growth, which had been diagnosed as an epithelioma of high malignancy, began to spread rapidly. On August 28, 1937, the growth was removed by surgical diathermy followed by electrocautery. This treatment left an ulcer 4.5 inches by 3.5 inches in diameter

and extending down to the deep fascia of the back. The lesion showed little tendency to heal and on September 6, 1937, the patient developed a severe secondary hemorrhage. Bleeding was stopped by the application of ligatures and packing. On September 9, 1937 treatment with cod liver oil-allantoin ointment was started. Improvement in the appearance of the wound was rapid. Figure 1 is a photograph of the lesion taken about a week after treatment was started and shows evidence of beginning granulation; the deep fascia is visible only in the center of the lesion. Healing continued without any secondary infection and on November 18, the entire ulcer was completely healed. No grafts were used to correct the skin defect. (Fig. 2.)

Fig. 5. Case IV. Showing appearance of infected foot in patient with diabetes several days after incision. Treatment with cod liver oil and allantoin had just been commenced.

Fig. 6. Case IV. Complete healing in eighteen days with insulin and the compound ointment.

Case II. I. G., white male, age 34, had a chronic infected ulcer of the left foot secondary to Buerger's disease (Fig. 3) of twelve years' duration. The right leg had been amputated above the knee. The left leg began to show symptoms about 1930, and the second, third, and fourth toes were amputated. A large, indolent ulcer of the dorsum of the foot developed subsequently. Treatment consisted of the following: left femoral sympathectomy in September 1930; intravenous sodium citrate solution for about six years; ultraviolet light treatment for three months in 1934; passive hyperemia, concomitantly with various salves locally for about one and one-half years during 1935 to 1937. Short wave diathermy and suction pressure therapy were also used for about six months. Under none of these various treatments was any improvement of the large infected ulcer on the dorsum of the left foot seen. The surface of the ulcer continued to remain infected, grayish in appearance, and extremely indolent. In July 1937 treatment with the cod liver oil-allantoin ointment was started. Since then the surface of the ulcer has cleared up considerably. The ulcer is now covered with fairly healthy granulations and appears clean. For the first time in seven years, there appears to be some attempt at epithelization at the edges of the ulcer. The deeper necrotic areas of the ulcer have disappeared and the entire ulcer is smaller and shallower,

and appears to be healing. (Fig. 4 taken December 20, 1937.)

Case III. I. G., white female, age 24, was admitted July 7, 1937 to the Mt. Sinai Hospital with a diagnosis of pregnancy of four and one-half months' duration, complicated by perforated appendicitis and localized peritonitis. Appendectomy was performed on the day of admission, the wound being drained with two Mikulicz drains. The wound become badly infected and was treated at first with urea crystals, but showed only a slight tendency toward granulation. On July 19, 1937 she developed a fecal fistula. The wound was packed with the special cod liver oil-allantoin ointment daily and showed a tendency to rapid granulation and healing. On July 26 the wound was clean and appeared much smaller. On August 10 the wound was practically completely healed and no longer discharging fecal material. On August 13 the patient was discharged with a completely healed wound.

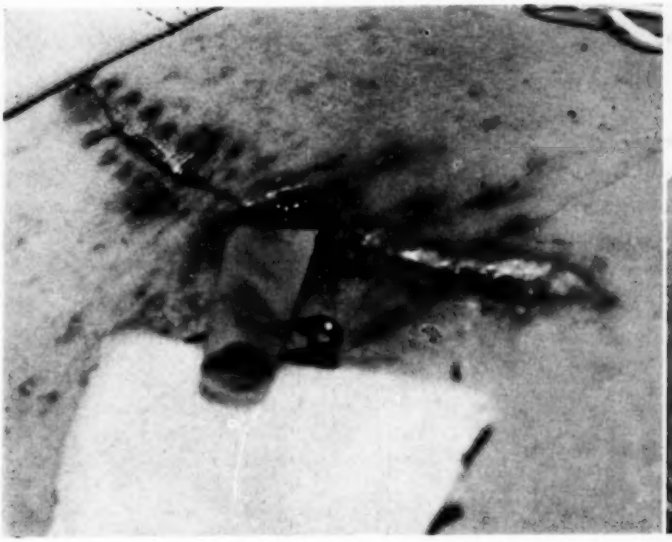


Fig. 7. Case v. Appearance of sinus following cholecystomy.

Fig. 8. Case v. Complete healing in ten days with cod liver oil-allantoin treatment.

Fig. 9. Iodine burn of the buttock three days after treatment with cod liver oil-allantoin ointment was instituted.

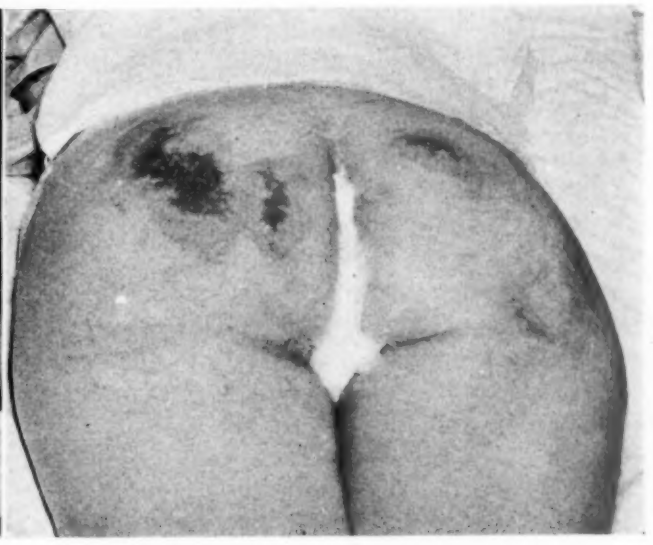


Fig. 10. Appearance of burned area three weeks after institution of treatment with cod liver oil-allantoin ointment. Epithelization has advanced remarkably.

Case IV. Mrs. S. C., age 40, had an infection of the small toe of the right foot with lymphangitis extending over the dorsum, following the trimming of a corn. Her blood sugar was 300 mg. per 100 c.c. Incision released a small amount of pus. The patient was treated with insulin and protamine zinc insulinate. Locally, daily dressings of cod liver oil-allantoin ointment were applied. Figure 5 shows the appearance of the foot several days after the institution of codalltoin treatment; Fig. 6 shows complete healing after treatment for eighteen days. Blood sugar was reduced to 167 mgs.

Case V. Mrs. M., age 56, had had a cholecystostomy for suppurative cholecystitis, and a sinus remained with drainage of a small amount of serum. (Fig. 7.) The sinus was surrounded by an ulcerative area whose border was lined with a few papules, and the condition was associated with pruritus. Daily dressings with cod liver oil-allantoin ointment were made. The ulcerative area filled in with pink granulation, and healing occurred in about ten days. (Fig. 8.)

SUMMARY

1. Allantoin, in the form of living maggots and extract of maggots, has been employed with remarkable success in the treatment of chronic cutaneous ulcerations, chronic osteomyelitis, severe burns and various dermatologic conditions.

2. Considerable evidence is available attesting to the beneficial effects of cod liver oil in the treatment of chronic non-healing wounds and chronic ulcerative affections.

3. The successful therapeutic use of cod liver oil and allantoin suggested a combination of these substances to obtain a double therapeutic action greater than the effect produced by either alone. It was found that such a combination with the addition of phenol was extremely effective in stimulating granulation tissue and encouraging healing in chronic ulcerative affections.

4. The ointment employed in this study contained 45 per cent cod liver oil, 2 per cent allantoin in a lanolin base with 0.5 per cent phenol.

5. Deep indolent ulcerative conditions and various cutaneous inflammatory affections respond promptly to applications of cod liver oil-allantoin ointment. The therapeutic procedure is simple and may be applied without any discomfort to the patient. It is tolerated better by the patient than the use of the actual maggots.

The preparation of cod liver oil-allantoin employed in this study is known as codalltoin and was made available through the courtesy of the Amfre Drug Company, New York City.

REFERENCES

1. Ayres, S., Anderson, N. P., and Talor G. M. Maggot therapy in dermatologic practice. Report of a case of chronic ulcerating granuloma of undetermined etiology in which maggot therapy was used. *J. Bone & Joint Surg.*, 13: 438 (July) 1931.
2. Baer, W. S. The treatment of chronic osteomyelitis with the maggot (Larva of the Blow-fly). *J. Bone & Joint Surg.*, 13: 438 (July) 1931.
3. Borovskaya, V. Lokale Anwendung des Lebertrans bei geschwierigen Lupusformen. *Sovet vrac. Gaz.*, 1904–1907, 1935.
4. Bramwell, William. The new cell proliferant. *Brit. M. J.*, 1: 12–13 (Jan. 6) 1912.
5. Brunsting, L. A., and Simonsen, Daisy G. Ulcers treated by the sulphydryl containing amino-acid cysteine. *J. A. M. A.*, 101: 1937–1940 (Dec.) 1933.
6. Davson, J. The tissue response to subcutaneous injection of cod liver oil. *Lancet*, 2: 737–738 (Sept. 26) 1936.
7. Drigalski, W. Zur Wirkung des Lebertrans auf Bakteriengemische insbesondere Erdbakterien und Sporenbildner. *Deutscb. med. Wcbnscbr.*, 62: 1005–1006, 1936.
8. Goldstein, H. I. Live maggots in the treatment of chronic osteomyelitis, tuberculous abscesses, discharging wounds, leg ulcers and discharging inoperable carcenomia. *Internat. Clin.*, 4: 269–282, 1932.
9. Goldstein, L., and Salzmann, H. A. Combined cod liver oil and allantoin therapy in chronic dermatologic affections. *Med. World*, in press.
10. Greenbaum, F. R. New granulation stimulating substance with especial emphasis on allantoin in ointment form. *Am. J. Surg.*, 34: 259–265 (Nov.) 1936.
11. Hammett, F. S., and Reimann, S. P. Cell proliferation response to sulphydryl in mammals. *J. Exper. Med.*, 50: 445–448 (Oct.) 1929.
12. Hayashi, S. Influence of cod liver oil upon bacteria and infected wounds. *J. Orient. Med.*, 24: 65, 1936.
13. Heald, C. B. Cod liver oil treatment of wound. *Lancet*, 2: 946 (Oct. 17) 1936.
14. Herlyn, K. E. Zur Lebertrangips-behandlung von Finger und Handverletzungen. *Brun's Beitr.*, 165: 278–282, 1937.

15. Holmes, A. D. External use of cod liver oil. *Indust. Med.*, 6: 77–83 (Feb.) 1937.
16. Kümmell, H., and Jensen, W. Beiträge zum Wirkungsmechanismus des Lebertranwindverbandes. *Deutscb. Ztscbr. f. Cbir.*, 248: 238–253, 1936.
17. Larrey, D. J. *Clin. Cbir.*, 1: 51–52, 1829.
18. Livingston, S. K. Maggot active principle. *Am. J. Surg.*, 35: 554–556 (March) 1937.
19. Livingston, S. K. Active principle of maggots with description of is clinical application in 567 cases. *J. Bone & Joint Surg.*, 18: 751–756 (July) 1936.
20. Livingston, S. K., and Prince, L. H. The treatment of chronic osteomyelitis with special reference to the use of the maggot active principle. *J. A. M. A.*, 98: 1143–1149, 1932.
21. Lähr, W. Die Behandlung grosser, flächenhafter Verbrennungen 1, 2, und 3. Grades mit Lebertran. *Cbirurg*, 6: 263–276, 1934.
22. Lundh, G. Experimentelle Untersuchungen der Wundheilung bei Tieren mit Lebertran. *Zentralbl. f. Cbir.*, 63: 2860–2868, 1936.
23. Macalister, C. J. A new cell proliferant: its clinical application in the treatment of ulcers. *Brit. M. J.*, 1: 10, 1912.
24. Macalister, C. J. The action of symphytum officinale. *Brit. M. J.*, 2: 702–704 (Sept. 21) 1912.
25. Pare, A. Les oeuvres d'Ambroise Pare, 1652.
26. Pyke, Magnus. Cod liver oil treatment of wounds. *Lancet*, 2: 1069 (Oct. 3) 1936.
27. Reimann, S. P. Use and reasons for the use of thiocresol to stimulate wound healing. *J. A. M. A.*, 94: 1369–1371 (May 3) 1930.
28. Ritter. Grundsätzliches zum Lebertran—Gipsverband. *Zentralbl. f. Cbir.*, 63: 1619–1621, 1936.
29. Robinson, William, and Norwood, V. H. The rôle of surgical maggots in the disinfection of osteomyelitis and other infected wounds. *J. Bone & Joint Surg.*, 15: 409–412 (April) 1933.
30. Robinson, William. Literature relating to the use of maggots in the treatment of suppurative infections. U. S. Dept. Agriculture, Bureau of Entomology. Circular E310 (Revised) June, 1934.
31. Robinson, William. Stimulation of healing in non-healing wounds. *J. Bone & Joint Surg.*, 17: 267–271, 1935.
32. Sandor, S. Vitamin A in the local treatment of wounds. *Lancet*, 2: 738–740 (Sept. 26) 1936.
33. Seiring, H. Fettsaüre Wirkung bei Lebertranverbänden. *München. med. Wchnschr.*, 83: 1632–1634, 1936.
34. Smelo, L. S. The problem of wound healing. *Arch. Surg.*, 33: 493–574, 1936.
35. Steel, J. P. The cod liver oil treatment of wounds. *Lancet*, 2: 290–292, 1935.
36. Stewart, M. A. A new treatment of osteomyelitis. *Surg., Gynec. & Obst.*, 58: 155–165 (Feb.) 1934.
37. Tejerina, Fotheringham W., and Gurruchaga, J. V. Allantoin in wounds. *Bol. y. trab. de la Soc. de cir. de Buenos Aires*, 20: 410–418 (June) 1936.
38. Tumanski, V. K., and Jaćević, I. A. Treatment of infected wounds with cod liver oil (Russian). *Klin. Med.*, 13: 1528–1537, 1935. Abst. *J. A. M. A.*, 106: 259, 1936.
39. Zachmann, J. C. Quoted by Goldstein, H. I. *Internat. Clin.*, 4: 269–282, 1932.

BENIGN TUMORS OF THE ILEOCECAL REGION*

WITH A SURVEY OF THE LITERATURE

MORRIS FREDERICK WIENER, M.D. AND SILIK H. POLAYES, M.D.

Clinical Assistant Surgeon, Beth Moses Hospital and Brooklyn Cancer Institute

Pathologist, Cumberland and Prospect Heights Hospitals

BROOKLYN, NEW YORK

THE following case reports and discussion are presented because they are concerned with two rare types of benign tumors found in the ileocolic region: one, an ileocecal submucous lipoma discovered at necropsy, and the other, a mesenteric fibroma found at laparotomy.

LIPOMA OF ILEOCECAL VALVE

C. K., a housewife 52 years of age, was admitted to the surgical service of Dr. Shatara of Cumberland Hospital, on September 20, 1931, suffering from severe abdominal pain.

Present Illness. The patient had had intense abdominal pain especially through the epigastrium, for three weeks prior to admission. In the last twenty-four hours this was associated with persistent vomiting.

Previous History. She had had rheumatic fever at the age of 20. Cholecystectomy was performed in December 1929, with partial relief of symptoms of abdominal pain, nausea and vomiting. At this time a palpable, smooth, mobile mass about 15 cm. in diameter was felt in the right lower quadrant by abdominal palpation and digital examination per rectum. The patient returned to the hospital four months later with a ventral incisional hernia which was treated conservatively.

Physical Examination. The patient was an obese white female, weighing 250 pounds, acutely ill, with a temperature of 100°F., pulse of 115 per minute, and rapid shallow respirations. The heart sounds were distant. The abdomen was obese and pendulous. A mass, which enlarged on straining, was found in the region of the abdominal scar. There was marked tenderness throughout the epigastrium. A diagnosis of incarcerated incisional hernia was made and an immediate operation was decided upon.

Operation. Excision of the midline scar (of previous operation) was made and the fascia split. The intestines and omentum immediately beneath were found to be adherent to the borders of the gaping peritoneum. The adhesions were freed and the defect in the peritoneum was closed in the usual manner.

Course. Following operation, the pulse was 150 per minute, respirations 40, and temperature 100.4°F. About nine hours after operation the patient became restless and irrational. There was marked perspiration. The radial pulse became imperceptible, the respirations rapid and shallow and the temperature made a terminal rise to 106.8°F., the patient expiring nineteen hours following the operation.

Post-Mortem Findings. The heart weighed 370 Gm., having considerable epicardial fat. The mitral valve was thickened along the line of closure. The anterior leaflet showed four minute, discrete, gray, firm, semitranslucent nodules, which, microscopically, consisted of hyalinized fibrous connective tissue. The spleen weighed 360 Gm. and was soft in consistency and red in color. The liver weighed 3,220 Gm., was yellow in color and microscopically showed marked fat replacement. The pancreas similarly showed a high degree of lipomatosis. The kidneys showed arteriosclerotic changes.

The *ileocecal valve* showed a large, soft, yellow, moderately lobulated mass measuring 5 × 4 × 3 cm. attached by a broad pedicle to the lower lip of the valvula coli. (Fig. 1.) This tumor was covered with normal mucosa and protruded into the lumen of the cecum. The cut surface presented a homogeneous, finely lobulated, yellow appearance. There was another similar ovoid mass, 2 cm. × 1 cm., proximal to the main tumor. Microscopically, the structure was typical of lipoma, containing sparse capillaries and supporting connective tissue. The overlying mucosa was normal and the muscularis mucosa was partly atrophied. The lymphatic spaces of the submucosa were dilated. The inner circular layer of the muscularis was not recognized at some points. The

* From the Department of Pathology of Cumberland Hospital.

external longitudinal muscle fibers were thin. The remainder of the gastrointestinal tract was normal.

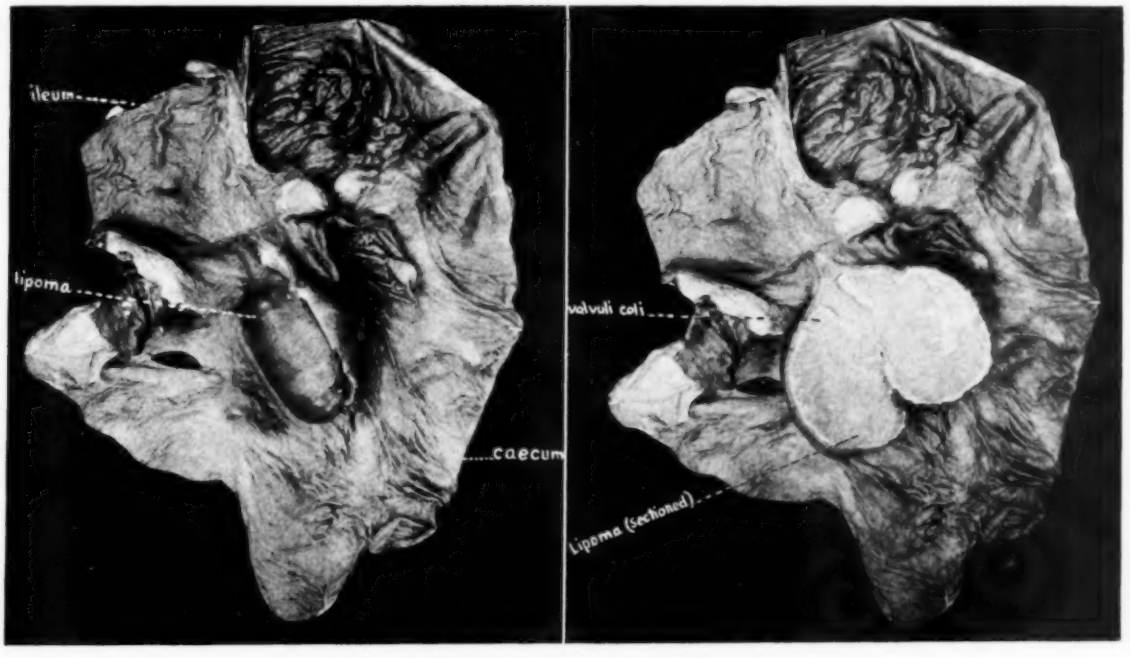


Fig. 1. Submucous lipoma of ileocolic valve.

The anatomic diagnoses were: obesity, status post-operative for incarcerated hernia, sclerosis of mitral valve (probably rheumatic), lipomatosis of liver and pancreas, nephrosclerosis, and lipoma of ileo-cecal valve. Death was probably due to post-operative shock in an obese female showing suggestive rheumatic infection of the heart.

MESENTERIC FIBROMA OF TERMINAL ILEUM

E. B., 42 years of age, colored laborer, was admitted to the surgical service of Dr. Chester Davidson, Cumberland Hospital, on August 6, 1935 at 2:00 A.M., complaining of severe lower abdominal pain.

Present Illness. The patient had had a dull, aching, suprapubic pain seven days before admission, aggravated a few days later by walking. He then went to bed where he was more comfortable when lying on the right side. During this period the appetite was good and the bowels regular. On August 5, the patient worked the entire day. At 2:00 A.M. of the next day, he was aroused from sleep by the increasing intensity of the suprapubic pain, which was continuous, aching, and radiated across to both lower quadrants. The last normal bowel movement occurred on Aug. 5. He vomited greenish material about eight hours after the onset of the acute pain. At this time, he developed epigastric pain after micturition.

Previous History. Epidemic parotitis with orchitis had occurred at the age of 32.

Physical Examination. The patient was a well developed and nourished colored male, 42 years old, acutely ill, with knees drawn up. There was exquisite tenderness in the lower quadrants with marked board-like rigidity. Rectal examination revealed similar tenderness, although more marked towards the right. The temperature was 102°F., pulse 84 per minute, respiratory rate 20 per minute, and blood pressure 126/83. The leucocyte count was 6,250 with 87 per cent polymorphonuclear neutrophiles and 13 per cent lymphocytes. The urine was normal. Blood Wassermann (alcoholic and cholesterin antigens) was one plus. A clinical diagnosis was made of perforated appendicitis with peritonitis and immediate surgery was recommended.

Operation. A lower right rectus incision, 20 cm. long was made. The muscles were split and the peritoneum opened. There was a large, ovoid, solid, tumor mass in the mesentery of the terminal ileum, the wall of which was "invaded" by the tumor and showed an area

of perforation. The peritoneum contained plastic lymph. The omentum was adherent to the mass at several points. There were no palpable nodes in the mesentery. There was a small quantity of free, purulent fluid present which showed B. coli on culture. The tumor and 15 cm. of the ileum were resected, the cut ends of the bowel were "purse-stringed" and inverted and a lateral anastomosis was performed. The abdomen was closed without drain in the usual manner. The postoperative clinical diagnosis was fibrosarcoma of the terminal ileum with perforation and localized peritonitis. Twenty-four hours after the operation the pulse was 128 per minute, temperature 105.2°F., respirations 12 per minute, and the blood pressure 80/64. There was improvement with intravenous and clyses of glucose. On the third postoperative day, the blood pressure was 124/80. On the sixth day, the abdomen became distended. On the seventh day, before death, the blood pressure rose to 150/100. The pulse rate increased to 150 per minute, and the temperature rose to 106°F. followed by exitus.

The post-mortem diagnoses were: generalized suppurative peritonitis, lobular pneumonia, focal necrosis of liver, surgical ileocecal anastomosis, anomalous double cystic duct and bilateral bifid ureters. (The examination of a specimen of blood taken at autopsy gave a three plus Wassermann reaction.) Death was due to postoperative suppurative peritonitis.

Pathologic Report of Surgically Removed Specimen. The specimen was an ovoid mass, 11 cm. in diameter, weighing 550 Gm. and to which was attached a segment of intestine 26 cm. in length. (Fig. 2.) The mass was firmly elastic and covered with a thin, pink-yellow glistening capsule which was continuous with the serosa of the intestine. At some points the mesenteric line was lost, due to the intimate union of the mass which appeared to merge with the wall of the gut. The intestinal mucosa showed an ulcerated area, 2 cm. in diameter with a sharp border and necrotic hemorrhagic base. This ulceration communicated with a small cystic cavity in the tumor. Microscopic examinations of sections from various areas of the mass showed a highly cellular edematous fibrous structure containing foci of leucocytic accumulations and congested vessels. The cyst wall was made up of fibrous tissue containing inflammatory cells. The adjacent intestine showed gangrenous change. A pathologic diagnosis of mesenteric fibroma and gangrene of ileum was made.

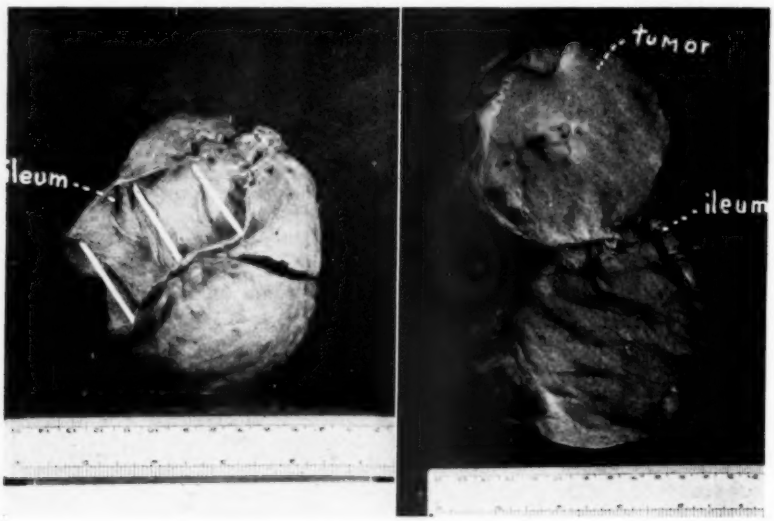


Fig. 2. Mesenteric fibroma.

INCIDENCE

Lipomata of the Gastrointestinal Tract. The anatomic distribution of all collected cases of gastrointestinal lipomata, totaling 259, may be classified as shown on p. 541.

Although the recent literature contains numerous references to enteric lipomas with involvement of every portion from esophagus to rectum, tumefaction of the ileocecal valve is singularly rare. Comfort, in his

Esophagus	2
Stomach	31
Duodenum	19
Small intestine	68
Ileocecal region	9
Ileocecal valve	3
Cecum	23
Colon	60
Sigmoid	21
Rectum	12
Incomplete accounts	11

series of 181 cases, describes only two in which the ileocecal valve was the seat of lipomatous growth, and Jefferson (1920) describes a fibroma of one of the lips of the ileocecal valve.*

Mesenteric Fibroma. Of 820,000 consecutive admissions at the Mayo Clinic, there were twenty-five cases of mesenteric tumors and only two were fibromata. Greer, in 1911, reviewed the literature of the preceding ninety years and collected reports of thirty-three cases. Harrison and Herzog, in 1897, reviewed some fifty-six cases, which, however, lacked histopathologic study. It is probable that most of the earlier cases reported represent curiosities of mesenteric growth and cannot be scientifically utilized for comparative study and classification. Proved cases of pure mesenteric fibromas have been reported by Royster (1911), Bevan (1918), Kyle (1921), Rawls (1923), De Courcy (1925), MacCauley (1925), Baldwin (1927), Grigorowsky (1928), Darnall (1928), Councellor (1931), Wakely (1931), Cox (1932), Summer (1932), McCalla (1932), and Weaver (1933).

Age. Case reports of mesenteric fibroma are found most often in adults. However, those found may have been present in childhood, without causing symptoms. Weaver, in 1933, reported a massive cellular fibroma of the mesocolon in a 10 year old colored female.

ETIOLOGY

Lipomata. There are large gaps in our present knowledge of the cause of tumors. There is increasing belief that several factors are involved, particularly of hormonal hereditary nature. The most that can now be definitely stated, is that a new growth is a result of developmental processes influenced by the genetic pattern of tissue development and by hormonal control through endocrine glands. The appearance of lipomatous tumors after puberty is one of many instances that suggests the influence of the gonadotropic secretion on tissue growth.

Mesenteric Fibromata. The origin of mesenteric new-growths is as shrouded in mystery as is the etiology of tumors elsewhere. Various theories have been suggested. The occurrence of mesenteric fibromata in the male readily weakens the hypothesis that these new growths result from detached uterine fibroids. The imperceptible merging of tumor mass and gut wall is the main factor to suggest origin from the intestinal wall. However, a more plausible and convincing explanation is that these benign neoplasms arise from segregated, mesoblastic remnants of the genital gland in the mesentery and sometimes referred to as origin in situ (from the connective tissue of the mesentery). The genital ridge develops lateral to the mesentery of the gut. From the common urogenital mesentery, the genital and Wolffian bodies derive their respective mesentery. The Wolffian duct is located in the medial margin of the genital ridge and dorsal to the müllerian duct. It is conceivable that portions of these fetal rests may migrate to the retroperitoneal tissue and invade the mesentery of the bowels and that under the influence of hereditary and hormonal factors these nuclei become the mesenteric tumors in postnatal life.

PATHOLOGIC ANATOMY

Lipomata. The lipomata may take the form of a small collection of fat cells in the submucosa, diffuse lipomatous infiltration, or discrete tumor mass. The fat cells are usually of the mature type, including the

* Since this survey was begun, the writer has observed another pedunculated submucous lipoma of the ileocecal valve, an accidental finding at autopsy.

microscopic varieties that are enclosed in the connective tissues of the submucosa. The amount and behavior of the connective tissue framework will determine the various types, i.e., fibrolipoma, liposarcoma, lipomyxoma, etc. As the tumors increase in size there is more definite demarcation between the submucosal connective tissue and the fat, the former developing into a capsule of variable thickness. Blood vessels, arterioles and capillaries are present and are normally developed. The mucosa is thinned and atrophic, although occasionally it is normal. The muscularis mucosa is not discernible in the larger tumors. Changes in the muscular layer are similarly dependent upon the size of the tumor mass as well as the degree of imposition by the tumor upon the blood supply of the gut wall.

Mesenteric Fibromata. Szenes collected sixty cases of mesenteric tumors which may be grouped according to the following simplified histologic basis:

Fibroma	25 (41 per cent)
Lipoma	7 (11 per cent)
Sarcoma	22 (39.5 per cent)
Lymphosarcoma	3 (4 per cent)
Endothelioma	2 (3 per cent)
Carcinoma	1 (1.5 per cent)

In this group 52 per cent are benign and 48 per cent are malignant. However, many of the latter are very likely not primary neoplasms of the mesentery. The histopathology of the primary tumors is probably determined at their early inception. Harris and Herzog (1897) refer to five cases of mesenteric tumors in children, all of which tumors were mixed types. Huge mesenteric fibromata are reported in adults, which suggest the long duration of these neoplasms. No changes occurred in the structural pattern of these new growths, the pure fibrous character of the tumors having persisted. The weight of the tumors may vary from 400 to 5,000 Gm. Baldwin (1927) described a mesenteric fibroma that weighed 11.3 Kg., which he claims is the largest ever to be recorded. Grossly, the appearance is that of an encapsulated, round, firm, mass, white in color, the cut surface being flat and intersected with glistening bands.

Microscopically, the fibroblast is the type of predominating cell. The proportion of cells to intercellular collagen fibers varies considerably. The type with abundant collagen has a firm consistency, while the highly cellular tumors are soft and more nearly approach sarcoma. The vessels are of the mature type and are usually sparse, although definitely increased in number in the growing tumors.

Most of the fibromata develop near the mesenteric attachment. This is significant in view of the frequent intimate adhesion to the gut wall and the ease with which the new growth usurps part of the enteric vascular supply. This relative ischemia of the gut wall decreases its tissue resistance and leads to inflammatory changes varying from a mild enteritis to gangrene, with ulceration and fistula into the tumor mass. These latter changes were observed in the writer's case report. The edema associated with acute inflammation may lead to rapid enlargement of the tumor, to which the inflammation may extend by way of the perivascular lymphatic channels. In such instances leucocytic infiltration around blood vessels of the tumor is frequently described together with the other usual inflammatory phenomena.

PATHOLOGIC PHYSIOLOGY

Lipomata. The size and location of the tumor will determine the time of onset of symptoms. These are probably due to: (1) interference with the intrinsic nervous mechanism of the gut wall, causing disordered peristaltic activity, which may explain some of the symptomatology in our case; (2) obstruction of the lumen; (3) intussusception; (4) ulceration of the lining mucosa or tumor itself; and (5) results of circulatory changes incidental to forceful peristalsis or intussusception.

Intussusception is the most important complication of submucous lipomata to demand surgical intervention. This grave

event most frequently occurs in tumors of the large bowel, sigmoid or descending colon, although the small intestine, particularly the ileum, is not uncommonly similarly involved. The mechanism and dynamics of intussusception are readily explained by the "terrain malidif" produced by the tumor lying in the intestinal canal. When the enteric mass acts as a foreign body, there result spasmodic contractions of the gut around it with inhibition and relaxation of the gut immediately distal to it. The conditions are now favorable for that act of peristaltic gymnastics whereby the contracted part is induced to slip into the dilated portion. To be sure, other factors may play some rôle in this phenomena, such as the perverted muscle action due to the presence of the tumor, the paralysis of the bowel itself and the traction due to the mere weight of the tumor. Antiperistalsis may account for the cases of retrograde intussusception.

Mesenteric Fibroma. The location and size of the fibroma in the mesentery are the essential factors in any disturbance of the normal intestinal physiology. A relatively small growth of the mesentery near its origin may produce angulation of the duodenojejunal junction causing a long train of symptoms so commonly associated with duodenal stasis, an effect similarly produced by an abnormally high insertion of the suspensory ligament of Treitz. The intestinal sympathetic nervous supply which follows the blood vessels may be compressed upon and produce abnormal muscle action of the gut wall. By mere enlargement the tumor mass encroaches upon the lumen and causes various degrees of intestinal obstruction. Constant pressure of the new growth against the gut wall leads to anatomic union between their opposing surfaces and brings peristaltic activity at this point to a standstill, such as most probably occurred in the case reported here.

DIAGNOSIS

Neither enteric lipomata or mesenteric fibromata give a well defined clinical picture. Their detection must always be a matter of difficulty and their presence is not suspected until laparotomy or necropsy. It is likely that such abdominal tumors may be present for years before producing symptoms. Pollosson and Rougemont have described the following syndrome which is peculiarly associated with submucous lipomata: The patient has several attacks of abdominal pain of such severity that he remembers each attack. He suffers from borborygmi and colicky pains which come on at regular intervals. There is complete abdominal relaxation with no distention and no rigidity. The pulse and temperature remain normal. If a tumor is felt at this stage, appendicitis or a similar inflammatory condition can be ruled out, as such conditions cannot form a swelling in a few hours. Examination of the urine may help to exclude renal colic. Abdominal palpation under an anesthetic may prove to be very expedient. To be sure, with the development of obstruction or intussusception, symptoms associated with these conditions will appear.

Wardill stresses the point that in operating for intussusception in subjects over 2 years of age, a careful examination should be made of the proximal healthy bowel for possible new growth. Hubner states that in all cases where an intestinal tumor was noted, an acute intussusception was the first sign of its existence. Frequently secondary changes, such as hemorrhage, infection, and edema, bring to light the existence of a hitherto clinically silent neoplasm. Where the invagination is high up there may be no blood present in the stool.

The diagnosis of mesenteric tumor should always be suggested when there is a mobile abdominal mass extrinsic to the gastrointestinal tract and where the horizontal mobility of the tumor is greater than the vertical. Osler mentions a tympanitic zone between the tumor and the pubis. Accurate diagnosis is not of great practical importance because the unmistakable tumor calls for surgical intervention.

PROGNOSIS

Prompt diagnosis of a tumor, regardless of the histologic structure, will tend to lower the mortality. Early surgical intervention reveals a smaller tumor mass with less likelihood of involvement of overlying intestine by intimate union with the gut wall or by usurping its blood supply. In the latter case the tumor mass may be successfully enucleated with however, subsequent development of a gangrenous process of the overlying intestine due to interference with its blood supply which was shared by both intestine and tumor. The dangers of hemorrhage, sepsis and shock are increased where intestinal resection is performed.

Generally, the mortality varies from 40 to 52 per cent, the higher percentage being associated with intestinal resections. Operative recoveries are reported by MacAuley (1925), Baldwin (1927), Fisher (1929), Councellor and Cox (1932), McCalla (1932), and Weaver (1933).

TREATMENT

Lipomata. In cases of intussusception, attempts at disinvagination should be made, and if this fails, resection either of the invaginated cylinder of gut or of the whole of the infected bowel should be done with entero-anastomosis (Leclerc). Where the condition is grave, exteriorization of the affected gut is urged. Since every tumor of the intestinal tract carries with it a threat of intussusception, the propriety of complete surgical removal will be generally admitted, unless there is a definite contraindication.

Mesenteric Fibroma. Mesenteric tumors are ideally treated by enucleation of the tumor mass with suture of the opening in the mesentery. This is possible only in the simplest cases. The indication for a systematic primary intestinal resection is often more real than is apparent, because of the common blood supply of the tumor and overlying intestine which renders removal of the tumor impossible without compromising the blood supply of the gut. Two factors must be considered in determining the question of intestinal resection, viz.: (1) union of tumor mass to gut wall; and (2) common blood supply of tumor and gut.

SUMMARY

1. Two cases representing two types of benign tumors of the right lower quadrant are presented with necropsy performed in each.

2. One is a submucous lipoma of the ileocecal valve and the other a fibroma of the mesentery of the terminal ileum.

3. A brief resumé is given of the incidence, etiology, pathologic anatomy, pathologic physiology, diagnosis, treatment and prognosis in each of the two conditions described.

REFERENCES

Baldwin, J. F. Huge fibroma of mesentery. *Am. J. Surg.*, 2: 160, 1927.

Bevan, A. D., and Gatewood, L. C. Mesenteric fibroma. *Surg. Clin. North America*, 4: 919, 1920.

Comfort, M. W. Submucous lipomata of gastrointestinal tract. *Surg., Gynec. & Obst.*, 52: 101, 1931.

Counseller, V. S., and Cox, F. W. Fibroma of the mesentery. *Surg. Clin. North America*, 12: 1033, 1932.

Darnall, W. E. Fibromata of the mesentery. *Ann. Surg.*, 87: 870, 1928.

Fisher, W. H. Mesenteric tumors. *Am. J. Surg.*, 7: 803, 1929.

Geschicter, C. H. Lipoid tumors. *Am. J. Cancer*, 21: 617, 1934.

Harris and Herzog. Solid mesenteric tumors. *Ann. Surg.*, 26: 66, 1897.

Kyle, H. G. Fibroma of mesentery. *Brit. J. Surg.*, 9: 295, 1921.

Jacoby, A. Fibroma of mesentery. *New York J. Med.*, 112: 66, 1920.

Jefferson, J. C. Fibroma of ileocaecal valve. *Brit. M. J.*, 2: 819, 1920.

Mac Auley, C. Solid tumors of the mesentery. *Irish J. M. Sc.*, p. 54, 1925.

Mc Calla, L. H. Fibroma of the mesentery. *J. South Carolina M. A.*, 28: 211, 1932.

Rankin, Fred, and Major, S. G. Tumors of the mesentery. *Surg., Gynec. & Obst.*, 54: 809, 1932.

Szenes, A. Über solide Mesenterialtumoren. *Deutsch. Ztschr. f. Chir.*, 144: 228, 1918.

Wakeley, C. P. Fibroma of the mesentery. *Brit. J. Surg.*, 18: 658, 1931.

Weaver, O. H. Fibroid tumor of the mesentery. *J. M. A. Georgia*, 22: 295, 1933.

THE PROBLEM OF BLEEDING PEPTIC ULCER*

CLINICAL ASPECTS AND SURGICAL INDICATIONS

LEON GOLDMAN, M.D.

Assistant Professor of Surgery, University of California Medical School

SAN FRANCISCO, CALIFORNIA

THERE is general agreement that patients with uncomplicated peptic ulcer should be treated conservatively, while those who develop perforation of the ulcer or organic pyloric obstruction, and those who prove to be refractory to medical treatment, should submit to surgery. The indications for surgery in patients who have had hemorrhage are not so clearly defined. There is, in fact, considerable controversy as to just what factors must be present before this complication requires surgery.

In an attempt to obtain a clearer conception of the indications for the surgical treatment of this type of patient, a study was made of 1,025 entries of 890 patients with peptic ulcer at the San Francisco Hospital, from January 1, 1928 to December 31, 1934.[1] Three hundred and forty-nine patients (38 per cent) entered the hospital because of gross hemorrhage from peptic ulcer or they developed this complication during their period of hospitalization. Of this number, thirty-nine (11.1 per cent) died of exsanguination, while an additional thirteen (3.9 per cent) died of conditions associated with the bleeding, such as perforation of the ulcer, pneumonia, postoperative complications, or cerebral or cardiac thrombosis, thereby bringing the total mortality of gross hemorrhage from peptic ulcer to 15 per cent.

The incidence of hemorrhage reached its peak during the fifth decade of life, and seven-tenths of the hemorrhages occurred after the age of 40 years. The highest mortality was between the ages of 40 and 70 years, when arteriosclerosis plays a part. Of those patients who died, approximately twice as many had duodenal ulcer as had gastric ulcer. This review further indicated that bleeding from a peptic ulcer is a poor prognostic sign and that the ulcer is likely not to remain healed permanently, probably because the majority of bleeding ulcers are on the posterior wall of the duodenum where chronicity is likely to ensue. Even though one would expect that the "death scare" of a severe gross hemorrhage would stimulate patients to follow medical instruction, nevertheless 40 per cent of these patients had had more than one gross hemorrhage. Contrary to the experience of others, the mortality from hemorrhage in our series increased with each recurrence. The pessimism of this situation may exist only in the management of patients with peptic ulcer in a city or charity hospital. The symptoms usually were relieved while these patients were in the hospital, even though penetration of the ulcer had occurred, by proper mental and physical rest as well as diet-alkali therapy. The discharge of these patients from the hospital to their more unfavorable surroundings where therapy could not be followed adequately, as well as their partaking of alcoholic beverages and excessive amounts of tobacco, often led to recurrence.

It is our belief that economic, social, and vocational, as well as constitutional or psychic factors, affect the incidence of recurrences and complications of peptic ulcer. A city hospital cares for varied groups of patients, many of whom are in poor social and economic circumstances and enter only because of complications of their ulcers. Many enter following dietary

* From the Department of Surgery, University of California Medical School (the University of California Service at the San Francisco Hospital) and from the Department of Public Health, San Francisco, California.

or alcoholic indiscretion; or their vocations are such that suitable therapeutic régimes cannot be followed. Only patients with complications of an emergency nature are admitted to the hospital. In this group, therefore, one may expect a higher incidence of and mortality from hemorrhage than are encountered in private hospitals.

Following, in general, Hinton's[2] plan of classifying his patients with bleeding peptic ulcer (in an analysis of 123 patients from his service at the Bellevue Hospital), we divided our cases into six groups for study. By such a study we hoped to evaluate each case—thereby suggesting the type of treatment—and at the same time, to analyze the type of patient who enters our city hospital.

Our six groups are composed of:

I. Patients with hemorrhage occurring during competent medical treatment.

II. Patients previously operated on for an acute perforation or chronic ulcer, but without hemorrhage for months or years following operation.

III. Patients who had been operated on previously for hemorrhage and who continued to have hemorrhage.

IV. Patients with severe hemorrhage but with either no history of peptic ulcer or a history of gastroduodenal symptoms of less than six weeks' duration; most of these patients did not know they had ulcers until hemorrhage occurred.

V. Patients admitted with hemorrhage and a long history of symptoms of ulcer, who were not following therapy at the time of onset of the hemorrhage.

VI. Patients with gross hemorrhage associated or coincidental with perforation.

Group I consisted of four patients, or 1.1 per cent of the 349 patients with hemorrhage. In the hospital records of only these four was there a statement that the patient was following proper or prescribed diet-alkali therapy at the time that the original hemorrhage took place. Many were under competent medical care, but deviation from their usual régime preceded the onset of bleeding. This finding is similar to that in perforation, in that the latter complication rarely occurs in patients who are following adequate diet-alkali therapy, and suggests that ulcers producing either hemorrhage or perforation are both active and spreading. Because of the location of bleeding ulcers on the posterior wall of the duodenum or the lesser curvature or posterior wall of the stomach, hemorrhage occurs as a result of erosion of the left gastric or superior pancreaticoduodenal artery, or branches of these arteries, which supply these areas. Rapidly penetrating ulcers on the anterior wall of the stomach or duodenum, especially in proximity to the pylorus, are likely to perforate into the free peritoneal cavity rather than to cause hemorrhage. An ulcer which causes gross hemorrhage, therefore, might be considered analagous to a perforated ulcer, but, because of anatomic differences, bleeding ensues. There were no deaths in Group I.

The small number of patients on adequate treatment at the time of hemorrhage suggests the absence of medical supervision or a lack of coöperation by the majority of the patients studied.

It is natural to conclude that, if a patient is following complete and adequate therapy during the same time that the hemorrhage occurs, surgical treatment, with removal of the ulcer if possible, should follow as soon as the patient's condition permits.

Sixteen per cent of Hinton's patients fell into Group I. The discrepancy noted in comparing our series with his (Table I) may

TABLE I
INCIDENCE OF BLEEDING PEPTIC ULCER BY CLASSIFIED GROUPS

Group	Percentage (San Francisco Hospital—349 Cases)	Percentage (Hinton's Cases 123, at Bellevue)
I	1.1	16.0
II	6.4	12.0
III	6.5	5.0
IV	21.4	18.0
V	62.4	49.0
VI	2.0	

result from the difference between the well developed out-patient or follow-up system used at his hospital and the lack of follow-up care or out-patient department at this hospital until very recently. Most of the patients who were discharged from this hospital previously were not heard from again, nor did they visit the other out-patient clinics in the city, as advised, until a complication developed.

Group II. Twenty-two patients (6.4 per cent of the entire number with hemorrhage) had been operated on previously for acute perforation or chronic ulcer, but had not bled until months or years later. Eleven, or 50 per cent, had had gastroenterostomy; eight had had perforation of an acute ulcer, with simple closure or pyloroplasty; one had had a Devine exclusion operation; one had had a pyloroplasty; and in one the type of operation was not known. Five, or 22.7 per cent, of the patients in Group II died. This evidence strongly suggests that indirect operations such as pyloroplasty or gastroenterostomy for duodenal ulcer may not protect the patient against subsequent hemorrhage or recurrence. Twelve per cent of Hinton's cases fell in Group II. He felt that, if the patient had had two hemorrhages, gastric resection with removal of the ulcer if possible, would usually become necessary. Our experience bears out his conclusion. The following is a synopsis of a typical case from Group II:

A man 63 years of age had a history of peptic ulcer dating back eighteen years. At the onset he was hospitalized and treated for duodenal ulcer, but after relief from his symptoms he was discharged from the hospital. Two years later a posterior gastrojejunostomy was done because of the recurrence of symptoms. Some fifteen years after his gastrojejunostomy, he entered this hospital because of recurrence of symptoms followed by hemorrhage. At this time a gastrointestinal *x*-ray series revealed a duodenal ulcer and a poorly functioning gastrojejunostomy. Following diet-alkali therapy, he again was relieved of symptoms and was discharged from the hospital with the advice that he follow a modified Sippy régime. He followed the therapy for only a short time, and about a year later he noted recurrence of symptoms. On the day of entry, for the first time, he vomited three pints of liquid and clotted blood, passed unchanged blood in the stool, and lost consciousness.

The skin and mucous membranes were moderately pale. The blood pressure was 90/50 and the pulse rate 88. Hemoglobin was 70 per cent, red blood cell count 4,080,000 and white blood cell count 14,300. He continued to have tarry stools, and the hemoglobin, red blood cell count, and blood pressure dropped. Six days later he had a sudden severe hemorrhage by rectum, and expired. Autopsy revealed an ulcer on the posterior wall of the duodenum, with a partially thrombosed arteriosclerotic vessel in the crater. This artery was a branch of the superior pancreaticoduodenal artery. There was no evidence of gastrojejunal ulceration.

Group III. Twenty-three, or 6.5 per cent of the entire number of patients, had gross hemorrhage after having been operated on for a previous hemorrhage. Nineteen of the twenty-three patients had had gastroenterostomy performed originally. Subsequent hemorrhage was treated by gastric resection in one patient and a Devine exclusion operation in another, with recurrences of the hemorrhages later. One had had a partial gastric resection for duodenal ulcer originally, and later bled. It is probable that the ulcer was not removed. In three patients, laparotomy had been done following hemorrhage from peptic ulcer, but the type of operation could not be determined. Three, or 13 per cent, of the patients in Group III died in the hospital. The percentage of patients in this group compares closely with that in Group II, suggesting that a recurrent ulcer may cause hemorrhage because of its location and proximity to a large blood vessel, even though hemorrhage had not been a feature before the original operation. It also suggests that gastroenterostomy may not protect a patient against the recurrence of a duodenal ulcer or against subsequent hemorrhage. In none of these cases was there evidence to suggest that the bleeding was from a marginal ulcer. Hinton reported that 5 per cent of his patients fell

into this group, most of whom had had gastroenterostomy.

Our policy regarding this group is similar to that followed in Group II because we believe that the incidence of recurrent hemorrhage will be lessened if the ulcer, with the distal part of the stomach, is resected. If the ulcer is on the posterior wall of the duodenum and infiltrates the pancreas, thereby making resection of the ulcer and the closure of the duodenal stump hazardous, the ulcer is left, its vessels are ligated and a subtotal gastric resection is done.

Group III is exemplified by the following report of one of our patients:

A man, 55 years of age, had had four entries into the hospital within two years. On his first entry a diagnosis of hemorrhage from a duodenal ulcer was made. After his condition had improved, an anterior gastroenterostomy with jejunojejunostomy was done. He was placed on a modified Sippy régime. Gastrointestinal *x*-ray series revealed a functioning stoma, and he was discharged from the hospital. He had several additional attacks of gastrointestinal bleeding, with tarry stools. For three months before his last (i.e., fifth) entry, he had noticed weakness, loss of weight, and epigastric pain occurring from two to three hours after meals, partially relieved by diet and alkali. Because of an attack of dizziness, hematemesis and melena, he entered the hospital.

The skin and mucous membranes were somewhat pale, the blood pressure was 130/80, and the pulse rate 92. There were no abdominal findings. Hemoglobin was 40 per cent and the red blood cell count was 1,190,000. Gastrointestinal *x*-ray series showed a functioning stoma without evidence of ulceration, but with a persistent defect in the duodenal cap. A diagnosis of recurrent duodenal ulcer with hemorrhage was made. The patient was placed on a modified Sippy régime, and later was discharged from the hospital, improved. A recurrence of his hemorrhage caused his death two months later at another hospital.

Group IV. Seventy-five patients, or 21.4 per cent, comprised this group, all of whom had either no history, or a very short one, of gastric symptoms. There were seventeen deaths, a mortality of 22.6 per cent, the diagnosis being confirmed by the coroner in the majority of cases. Hinton reported twenty-two cases in Group IV, an incidence of 18 per cent in the entire series, with a mortality of 53 per cent.[3] Many of our patients in this group died within a few hours after entry and most of them were in severe shock when seen first. Almost all of them were in the arteriosclerotic age. They were in such poor general condition that only a brief history could be obtained. In certain instances complete histories were obtained later. In some, symptoms of ulcer were found to have existed years previously, thereby placing these patients in Group V.

It is in Group IV that the diagnosis of peptic ulcer as the cause of the hemorrhage must be differentiated from hepatic cirrhosis, splenic anemia, gastric carcinoma, blood dyscrasia, toxemia with acute gastroduodenal ulcerations, and a few other rare conditions. The absence of any antecedent history of peptic ulcer makes the diagnosis difficult except for the fact that peptic ulcer is by far the most common cause of hematemesis. Bulmer[4] reported gastroduodenal ulcer as the cause of hematemesis in 88 per cent of 649 cases of hematemesis, so that, if one can eliminate hepatic cirrhosis and splenic anemia or other diseases of the blood, the percentage of diagnostic error will be very small.

It is for this group of patients more than for any other that donors should be available immediately and transfusions of from 250 to 300 c.c. of citrated blood be administered slowly. Procrastination may prove tragic because death may suddenly take place. Immediate surgery is likely to be futile during the acute bleeding phase in this particular group because of the poor general condition of the patient. Most of these patients have not been aware of their ulcers and, if they recover from the hemorrhage, they should be given a trial on medical treatment; usually they improve.

The following report is typical of patients in this group:

A man 68 years of age suddenly collapsed on the floor two days before entry to the hospital; he lost consciousness and passed a large black stool. The next day the same thing occurred, associated with the vomiting of a large amount of blood. Two weeks before, he had had rather sharp epigastric pain occurring from one-half to one hour after meals, relieved by food and soda. He had had no previous history of peptic ulcer or hemorrhage.

The patient was pale and hyperpneic, with a blood pressure of 80/60. He appeared rather anxious and was thrashing about. The hemoglobin was 35 per cent and the red blood cell count 1,200,000. He continued to vomit bright red blood and had a large involuntary black stool. He died three hours after entry, before a blood donor was procured, and the coroner's report noted the presence of a duodenal ulcer with an open sclerotic artery in the crater.

Group V. This group is the largest and comprises 218 patients, or 62.4 per cent of those with hemorrhage. There were twenty-five deaths, a mortality of 11.4 per cent. This group was also largest in Hinton's series, making up 49 per cent of his patients with hemorrhage, with a mortality of 10.5 per cent.

All of our patients in this group had had symptoms for more than six weeks before entry, most of them knew that they had peptic ulcer, and many had had several hospital entries. Either lack of medical supervision or of coöperation by the patient probably explains the size of this group. It illustrates the necessity for adequate therapy and close observation over a period of many years. Of the 135 patients reëntering this hospital during the seven-year period, the majority fall into this group. It is probable that many left this hospital and entered others when hemorrhage or recurrence took place. An index of their lack of coöperation is the fact that, of 193 patients in this group discharged from the hospital, thirty-six, or 18.6 per cent, signed releases, indicating that they felt further therapy to be unnecessary. The prompt relief of their symptoms brought about by rest in bed, hospitalization, diet-alkali therapy, and antispasmodics gave them such a false sense of security that 18.6 per cent of these patients left the hospital contrary to the advice of the medical staff. The mortality and the economic loss to the patient and the community can be curtailed as soon as patients are educated to know that they will be subject to recurrences as long as they live, and that gross hemorrhage, as well as perforation, is rare in patients following adequate therapy.

If arteriosclerotic patients in this group, over 40 years of age, continue to bleed under adequate medical therapy, including transfusions, surgery should be considered in order to prevent death from exsanguination. Because we know that patients in this group are likely to continue in their own way after being on an ulcer régime for a short time after discharge from the hospital, we advocate surgery after the occurrence of two hemorrhages.

Illustrative of this group is the following rather typical record:

A patient, 28 years of age, six years before entry to the San Francisco Hospital, developed epigastric burning which occurred two hours after meals and was relieved by food and soda. A physician diagnosed a peptic ulcer and put him on a diet-alkali régime. Shortly afterward, hematemesis of one pint of dark blood, followed by tarry stools and dizziness occurred. He was hospitalized and a diagnosis of ulcer was made by *x*-ray. He remained on a diet for nine months, then deviated from it and had another hemorrhage. At this time he entered Boston City Hospital, where *x*-ray examination revealed a duodenal ulcer. He continued to have periods of remissions and exacerbations, with a recurrence of hemorrhage three years before entry to our hospital. He followed a modified Sippy diet of his own selection for some time. Two months before entry his pain recurred and because of hemorrhage, he finally entered the hospital. Examination of the abdomen was negative except for slight tenderness to the right of and above the umbilicus. The hemoglobin was 56 per cent and the red blood cell count 3,050,000. Tarry stools were present. Gastrointestinal *x*-ray series revealed a duodenal ulcer. Under a Sippy régime, his bleeding stopped and he was relieved of symp-

toms. At the patient's request, he was later discharged from the hospital.

Group VI. This group includes seven patients who had two serious, dramatic complications of peptic ulcer, namely perforation and hemorrhage, during the same hospital entry. This comprised an incidence of 2 per cent of the whole series, and there were six deaths—a mortality of 85.7 per cent. Only two of these patients were operated on, the others being almost moribund on entry. Although there is a common impression that bleeding ulcers do not perforate and perforating ulcers do not bleed, these sequences occurred in seven patients. In cases of perforation, the duodenal ulcer usually is on the anterior wall and many times is associated with a "kissing" ulcer of the posterior wall which may be the site of a hemorrhage from the superior pancreaticoduodenal artery. This has occurred postoperatively after the site of perforation has been closed by suture. Patients may have a gastric as well as a duodenal ulcer, with hemorrhage of one and perforation of the other. The other type is the bleeding ulcer located in the posterior wall of the duodenum, which, because of its active progression, spreads and involves the intraperitoneal anterior wall, through which perforation occurs. Occasionally multiple, rather acute gastric ulcers form during the progress of an acute infection and present complications of hemorrhage and perforation.

This group is exemplified by the following record:

A man, 59 years of age, entered the hospital complaining of gastric distress following meals, of a year's duration. Two days before entry, he developed rather sudden pain in the epigastrium, which became generalized. The pain gradually disappeared the next day, but he entered the hospital. At that time his epigastrium was rigid and there were no palpable masses. The hemoglobin was 88 per cent and the red blood cell count was 4,100,000; the white blood cell count was 15,000. Immediately following entry, he developed severe hematemesis and melena. Because of his poor condition, he was treated conservatively. His bleeding continued, however, and he expired three days later. At autopsy, an ulcer of the lesser curvature of the stomach was found, measuring 2 cm. in diameter. There was a large vessel projecting into its lumen. In the same region, there were also multiple small ulcers, one of which had perforated, causing a localized peritonitis.

SUMMARY

1. In a city hospital where patients with peptic ulcer enter only because of some serious complication of ulcer, there is a high incidence of hemorrhage and resultant mortality. In our experience, this is greatest during the arteriosclerotic age. The mortality rises with each hemorrhage.

2. This study suggests that the occurrence of hemorrhage, similar to that of perforation, is rare in patients following prescribed or proper diet-alkali therapy. In the majority of cases, dietary or alcoholic indiscretion precedes the onset of these complications.

3. Although one must treat each patient according to the merits of his individual case, based on the age of the patient, the presence of arteriosclerosis, the location of the ulcer and the past history, the data collected suggest that indications for surgery may be present in each clinical group as follows:

(a) In the few patients who are following adequate treatment at the time of the onset of the hemorrhage (Group I), surgical treatment should be considered during the quiescent stage.

(b) If surgery is performed for the bleeding type of ulcer, direct attack on the ulcer with its removal, in addition to subtotal gastrectomy, is the procedure of choice and is more likely to reduce the incidence of hemorrhage after surgery. The study of Groups II and III suggests that gastrojejunostomy may not protect against the recurrence of or subsequent hemorrhage from a duodenal ulcer. Thirty patients of this series had gross hemorrhage from ulcer after gastroenterostomy.

(c) Patients with no history of peptic ulcer, or a very short history, who suddenly bleed (Group IV), have the poorest prognosis for life in the entire series. Transfusions are important in the treatment of this particular group. Surgery is not feasible in the acute stage because of the patient's poor general condition. After the hemorrhage has ceased, these patients usually do well on medical treatment.

(d) Sixty-two per cent of the patients with hemorrhage have had the symptoms of ulcer for many years and, in our experience, represent an uncoöperative group (Group V). If, during the acute bleeding phase, hemorrhage keeps recurring while the patient is under medical treatment, with transfusions, immediate surgery should be considered. After the occurrence of two hemorrhages, surgery is indicated during a quiescent period because it is very likely that these patients will not follow prescribed therapy over a long period of time, and the hemorrhage will recur. Conservative treatment is indicated in those patients below the age of forty years, without arteriosclerosis.

4. Hemorrhage and perforation occasionally are associated or coincidental; the prognosis is poor.

CONCLUSION

Indications for the surgical treatment of peptic ulcer with gross hemorrhage should be based on the age of the patient, the number of hemorrhages, the presence of arteriosclerosis and a study of the clinical aspects of each case.

REFERENCES

1. Goldman, Leon. Gross hemorrhage from peptic ulcer; its morbidity, mortality and treatment. *J. A. M. A.*, 107: 1537–1541 (Nov. 7) 1936.
2. Hinton, J. Wm. Significance of gross hemorrhage in peptic ulcer. *Am. J. Surg.*, 33: 180–182 (Aug.) 1936.
3. Hinton, J. Wm. Massive hemorrhage in peptic ulcer. *Ann. Surg.*, 101: 856–862 (March) 1935.
4. Bulmer, Ernest. Mortality from hematemesis. *Lancet*, 2: 720–722 (Oct. 1) 1932.

CHOLECYSTECTOMY (SUBSEROUS) WITH CYSTIC DUCT DRAINAGE AND CHOLANGIOGRAPHY

W. O. Sweek, M.D., F.A.C.S., T. A. Hartgraves, M.D. and H. G. Williams, B.S., M.D.

Lois Grunow Memorial Clinic

PHOENIX, ARIZONA

In this paper we are reporting a series of 213 cases of chronic cholecystitis, with and without stones, in which the gall-bladder has been removed subserously and cystic duct drainage instituted. One death occurred in the series.

For the past fourteen years we have been doing all cholecystectomies subserously where possible and leaving a tube firmly tied in the cystic duct or in the serous sac after removal of the gall-bladder. This tube remains until the ligature dissolves. We have had three cases in which the tube was pulled out by the patient in forty-eight to sixty hours, but no harm has ever resulted. The average time for the ligature to dissolve or the stump of the duct to separate is approximately six days.

The gall-bladder is removed subserously and a No. 12 or 14 French catheter is anchored in the cystic duct with No. 1 plain catgut.

Subserous resection was first reported by Doyen,[1] but no one advocated cystic duct drainage as a routine procedure until 1926.[2] Albert Ochsner[3] of Chicago suggested it, but failed to advocate fixing a tube in the cystic duct for the purpose of washing out the common duct and facilitating bile drainage of the liver and biliary tree. In 1921, Reid and Halstead[4] advocated draining the common duct through the stump of the cystic duct, but their procedure was different from the one we use.

Drainage of the bile ducts tends to keep down bile pressure and prevent leakage through bile capillaries from raw surfaces left by dissection, thus acting also as a safety valve. If lipiodol is injected into the biliary system of a dog following the stripping of the gall-bladder from the liver surface, it is frequently demonstrated to the eye; if the abdomen is closed, it is clearly visible on the *x*-ray film. (Figs. 7 and 8.)

By carefully separating the serosa from the gall-bladder as far down as is easily accomplished and loosening and drawing out the cystic duct, the common duct can be brought into view. If there is a stricture or a small stone in the cystic duct, the latter is more easily broken or pulled off, but even should a break occur from traction, no harm is done. As much as the cystic duct as possible is cut away as close to the common duct as 1 cm. or more. A large tube is then placed in the serous sac as far down as it will go and ligated securely in place by passing a No. 1 chromic gut suture around the serous coat of the gall-bladder, a mere pocket, since the gall-bladder has been removed. Drainage is copious for two to five days and the patient seems as well off as in cases where the tube is fastened in the cystic duct. If there is no evidence of stone or stricture, the common duct should not be opened.

When the cystic duct tube is fastened in place, 20 to 40 c.c. of warm magnesium sulphate solution is slowly injected into the bile ducts through the tube fastened in the cystic duct. If there is common duct obstruction from stone or stricture, the patient will complain of pain when 3 to 5 c.c. of the solution has been injected. This test, obviously, is of value only in cases operated on under local anesthesia.

Ninety per cent of our gall-bladder and duct surgery is done under local anesthesia (spinal is never used). A general anesthetic is employed only in rare cases of multiple and firm adhesions with the stone imbedded

in the ampulla, or in extremely nervous people with adhesions and stones requiring transduodenal removal. The technique of local anesthesia is easily acquired. Farr is still the authority regarding the procedure and nothing worth-while has come to our attention since his last authoritative text.[5] In our series of 213 cases, 187 were done under local anesthesia.

Fig. 1. First film, Case 1, made with the patient in supine position. This shows very plainly an indentation at the bottom, clearly indicating an obstructive object in the common duct with at least part of the surface oval, which would be interpreted as a large stone in the common duct near the duodenum.

Fig. 2. *X*-ray of same patient taken in the erect position. The small tube can be seen and the point where it is attached to the duct. These two films were taken five days after the operation.

When the gall-bladder is grasped in a clamp and brought up, a needle is carefully introduced beneath the serosa and from 40 to 60 c.c. of ¾ per cent novocaine is introduced. This serves two purposes. It separates the serosa from the gall-bladder and reduces the abdominal reflex tension. The gall-bladder has no sensory fibers, and if traction is not exerted the patient will not complain. After the injection is made, steady traction will cause the diaphragm to exert an almost steady pressure sufficient to push the liver and gall-bladder downward and afford adequate exposure for good work.

Contraction of the sphincter of Oddi (or the muscular coats of the duodenum) can be relaxed by the introduction of magnesium sulphate. When the solution is first introduced, there is marked pylorospasm; the pyloric sphincter can be seen to contract and the entire area gives an impression of hard muscular contraction. Usually the patient complains of pain when the contraction starts, but this is immediately followed by a free flow of bile back through the cystic duct tube. The pyloric sphincter relaxes in a few minutes and in many cases the patient vomits bile. The output of bile is often so profuse that it forms a thin

stream from the cystic duct tube for several minutes.

Thirty-four per cent of our cases have shown stones in the gall-bladder and ducts, 8 per cent having common duct stones.

The use of routine cystic duct drainage affords a simple and easy method of making a radiograph of the ducts after removal of stones and any other pathologic conditions requiring surgery. After passing magnesium sulphate solution or sterile physiologic saline through the tube fastened in the cystic duct to detect leaking points, lipiodol is introduced in the same manner. A sterile sheet is spread over the operative field and the portable *x*-ray is used to take the necessary films. A ten-minute period is usually sufficient to develop the film and return it.

Cutting down on stones in the common duct is a hazardous procedure for the most experienced surgeon and should never be attempted by the casual operator. We cannot agree with some of our colleagues (e.g., Walters[6]) that "if a patient can stand a gall-bladder operation of any kind he can stand to have the common duct explored."

We have occasionally removed the gall bladder, collected the bile through a tube, and reintroduced it by way of a duodenal tube. This procedure enables the patient to regain weight, brings the icteric index down to normal and converts a poor or fair to a good risk. We report one such case as a typical example:

Fig. 3. Case 11. Taken with the patient on the operating table. Shows the lipiodol filling the common and hepatic ducts and the lower portion of the gall-bladder below the ligature.

Case 1. Mrs. B. (Reg. No. 26180) presented herself with a history of intermittent jaundice, chills and fever of eight months' duration. She gave a history of having had a cholecystotomy eight years ago, but no stones were found at that operation. She had lost 47 pounds since the onset of her illness. The icteric index was 60.

On October 10, 1936, cholecystectomy was performed, and stones were found in the gall-bladder and common duct. The patient complained of pain when only a few c.c. of magnesium sulphate were injected through the cystic duct tube, and we were positive that a stone had lodged in the common duct. Her condition would not permit opening of the common duct, however. Improvement was rapid and she was sent home. She collected the bile from the cystic duct tube and took it each day through a duodenal tube. A cholangiogram

at this time showed an obstruction of the common duct, probably stone. (Figs. 1 and 2.)

A third operation was done on November 10, 1936, at which a soft stone about 1.5 cm. in diameter was removed transduodenally from the common duct at the ampulla of Vater. She made an uneventful convalescence, and when last seen (December 15, 1936) had gained 15 pounds and was in excellent health.

Fig. 4. Shows the lipiodol outside of the biliary tree in the duodenum as a large round shadow on the plate. This patient had a typical history of stones, including chills, jaundice, etc., but at operation no stones were found. No *x*-ray was made prior to operation. The case was an acute obstruction and a very typical one. If there was a stone present it was washed out by the pressure of the lipiodol and does not show on the film.

Had operative interference been carried out in this case to the completion of the operation, it is our opinion that this patient would have died on the table or shortly afterward.

Cases of intermittent fever and jaundice are usually indicative of common duct stone, although the stones are frequently observed in the cystic and hepatic ducts or in the gall-bladder instead. However, there are cases, such as Case II, in which, in spite of such symptoms and signs, no stone is present. Our records show several such cases.

Case II. W. T. B. (Reg. No. 48956) presented himself with a history of recurrent attacks of epigastric discomfort, colic, fever, and occasional jaundice. The icteric index at this time was 12, the blood count slightly elevated, and the urine negative. A preoperative diagnosis of cholelithiasis and stones in the common duct was made.

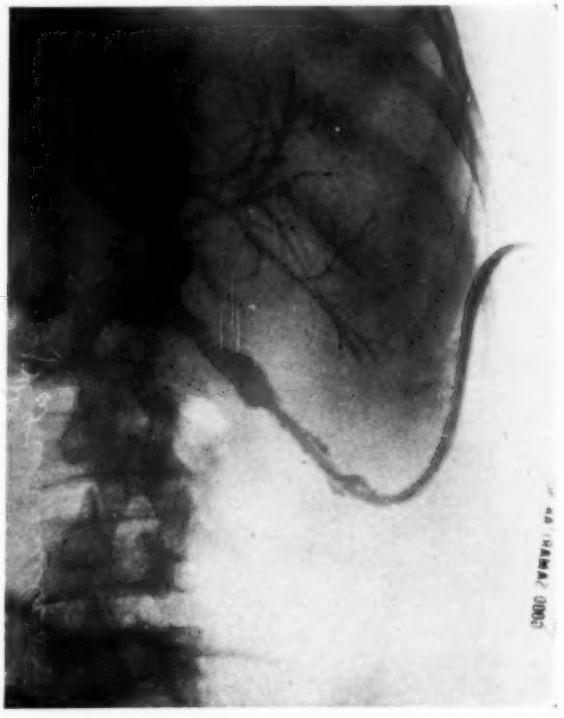


Fig. 5. Case III. Radiograph showing stone in common duct producing complete obstruction. Note concavity of lipiodol shadow at point of obstruction.

Subserosal removal of the gall-bladder was done, and lipiodol was injected into the biliary tract through a catheter in the gall-bladder while patient was on the operating table. (Fig. 3.) Another radiograph, five minutes later, showed lipiodol in the duodenum, with no indication of stones in the common or hepatic duct. (Fig. 4.) No stones were found and the common duct was not opened. The patient made an uneventful recovery and is in excellent health at the present time.

Case III. Mr. J. A., age 50 (Reg. No. 27117), had a history of gall-bladder trouble over a period of several years. He had had hypodermics on two occasions for epigastric pain, apparently gall-bladder colic, but had never been jaundiced until the onset of his present illness. Two weeks before his entrance into hospital, the patient developed jaundice which gradually increased, accompanied by abdominal pain, fever and two slight chills. Preopera-

tive treatment in the hospital did not lower his icteric index, which at the time of operation was 75.

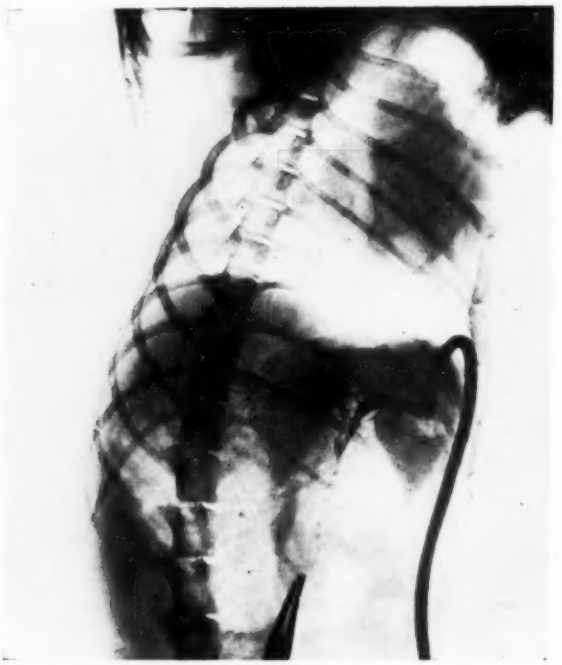


Fig. 6. Dog #8. The tube and the duct were fastened together by passing the tube into the gall-bladder as far down as it would go and then ligating it. The common duct was filled with small stones by passing them in through the ampulla of Vater. The common duct was occluded at the duodenum by grasping in the forceps, and the gall-bladder and duct were pulled upward, leaving the common duct in the straight line. The lipiodol is seen as round, shot-like bodies between the irregular stones.

A cholecystectomy was done January 8, 1937 and cystic duct drainage carried out. Many stones were present in the gall-bladder, and a cholangiogram on the operating table showed an obstruction of the common duct. However, the patient's condition would not allow further operative procedures.

The material contained in the gall-bladder was clear, with no evidence of bile pigments. Bile was flowing from the cystic duct tube before the patient left the operating room, and approximately 200 c.c. drained during the first three postoperative days, after which there was no further flow. Within thirty minutes time following the injection of 20 c.c. of whole blood into the liver, bile began to flow copiously from the cystic duct tube and continued to flow thereafter. Practically all the bile the liver secreted (about 900 c.c. per day) came out through the cystic duct tube. The patient preferred to drink the bile recovered from the cystic duct tube rather than swallow the duodenal tube; this was continued until the icteric index was 10.

A second operation was performed February 9, 1937, at which time a stone impacted at the ampulla of Vater (Fig. 5) was removed transduodenally. He made an uneventful recovery except that bile did not enter the duodenum until two weeks later due to swelling around the operative site. As shown by subsequent cholangiograms, this partial occlusion has since disappeared and the patient has gained 12 pounds in weight.

We have reported only these three cases since they are clear cut and illustrative of the value of cholangiography.

Cholangiography is a clinical aid not only at the operating table but also postoperatively in determining the cause of persistent biliary fistulae. It is our opinion that this diagnostic test should become extremely useful and prevent many damaging and fatal procedures in gall-bladder and hepatic duct surgery.[7]

By the practice of cystic duct drainage and the routine washing of the ducts with warm saline or magnesium sulphate solution we have not, to our knowledge, left unrecognized any obstruction in the common duct. Stones in the cystic and common ducts below a stricture of the cystic duct are frequent enough to give abdominal surgeons cause for worry. When we cannot pass fluid freely into the duodenum through the tube, we proceed immediately to extend the operative procedure if the patient's condition will permit. The injection of lipiodol into the cystic duct drain tube makes possible location of the stricture or stone. If the patient's condition warrants it, the operation is completed.

The free passage of bile or magnesium sulphate into the duodenum is not proof in all cases that small stones do not remain in the common duct; the flow of bile past such stones is too common and well known to need discussion. Cholangiography with lipiodol will, however, detect even such small stones.

The detection in the common duct of stones which do not obstruct and cannot be palpated is at present, therefore, possible by two methods only. One is to open the common duct in all stone cases, but this may fail in cases where the stone is dislodged backward into the hepatic ducts. As a general rule, the surgeon opens the common duct only if stones have been found in the cystic duct or the gall-bladder. As mentioned before, small stones may have passed into the common and hepatic ducts and a stricture may have formed above in the cystic duct. This makes it mandatory for the surgeon to open and explore the common duct in all cases with a history of intermittent fever in gall-bladder disease, all cases of stone, and in all cases of cystic duct obstruction interfering with the free passage of fluid through the bile ducts to the duodenum.

The second method of detection, cholangiography, will obviate the necessity of opening the common duct in many cases. It will also detect stones in the hepatic ducts that might be easily overlooked if merely a direct exploratory incision of the common duct were made.

Fig. 7. Dog #3. Escape of lipiodol from surface of liver slightly torn by removal of gall-bladder.

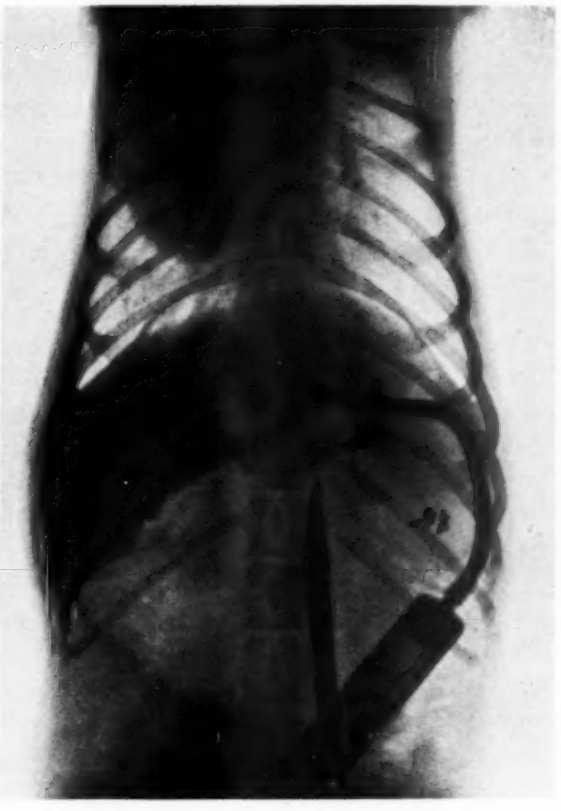


Fig. 8. Radiograph of same dog as in Figure 7 a few minutes later. Note lipiodol at site of gall-bladder and, lower down, cluster of droplets that have escaped from torn surface of liver.

In order to devise a method of demonstrating the presence of stone in the common and hepatic ducts, we proceeded to perform a series of experiments on dogs. We introduced stones of various sizes into the bile ducts, ligated the common duct near the duodenum, and then introduced a catheter into the lower segment of the gall-bladder. The bile ducts were clearly outlined by the slow injection of 3 to 5 c.c. of lipiodol, small definite areas of lessened opacity indicating presence of stones. (Figs. 6, 7 and 8.) We prefer lipiodol because of the density of the shadow and also for the very important fact that it will not injure the patient should it enter

a vein or escape through bile capillaries or aberrant ducts[8] into the peritoneal cavity. Should it escape, it will appear as shot-like bodies, warning the surgeon that there is bile leakage due to the stripping of the gall-bladder from its bed or to a damaged duct. (Figs. 7 and 8.) The more dense the medium, the clearer the outline of the stone will be, except in the case of such stones as are opaque to *x*-ray.

A word on the reduction of unnecessary traumatization of the hilus of the liver, may not be out of place. Dogs subjected to operation on the gall-bladder and ducts seldom die from these ordinary procedures. However, dogs subjected to excessive trauma by dragging, crushing or injection of irritating substances, such as strong salt solution, 90 per cent alcohol or 10 per cent sodium hydroxide, frequently die in shock. The experiment is done by injecting these substances along the cystic and common ducts so that the nerves to the liver are damaged. Clamping and crushing of the vessels surrounding the hilus of the liver will frequently produce shock in a dog. Dragging on the liver and rotating it, as is frequently done in human cases, will produce death in some dogs and will always produce symptoms of shock.

In human cases the excess trauma produces many of the postoperative complications, such as vomiting, etc., and materially increases the mortality and morbidity. In this connection, we may note that thickening of the common duct is not a reason for opening it, since all major bile ducts and the gall-bladder itself are usually altered in cases of cholecystitis. There is also evidence of inflammatory processes invading the hilus of the liver extending along the ducts, and hepatitis of variable degree is invariably present.

Until further evidence is presented, dysfunction of the gall-bladder, stones, strictures or pancreatitis, and the clinical symptoms indicating disease of the liver and gall-bladder will, to a great extent, remain in the realm of surgery. By this statement we do not imply that medical treatment does not enter into the situation. Close coöperation between internist and surgeon is of paramount importance to the patient, and it is our practice to coöperate with the internist in all cases possible. Many patients operated on for gall-bladder disease require careful and intelligent treatment by the internist after operation. For example, where the pancreas is involved sufficiently to be detectable by palpation at operation, the patient improves slowly and in some cases does not reach the maximum of improvement in less than two years. Medical and surgical research into this almost virgin field should proceed hand in hand; cöoperative effort may help the vast number of people suffering from the many symptoms resulting from liver pathology.

Concentration on the pathology involving the liver, pancreas and their ducts should not blind us to the possible contributing causes of these pathologic and frequently fatal conditions. The focal infection hypothesis should be earnestly and conscientiously considered, although it is often erroneous. Frequently we see patients with advanced hepatic disease consulting one specialist after another until all of the possible minor areas of infection have been removed. Operations on teeth, tonsils, and appendix, and all varieties of sinus operations, etc., are usually done first. In the case of female patients the gynecologist does various repairs, removals, etc. A long series of quacks has a chance, and then finally, the internist and surgeon have an opportunity to examine the patient. Most of the *causes* of the liver pathology have probably been removed, but either they were removed too late, or the "wrong cause" was removed.

We believe that patients with long-standing abdominal symptoms, whether esophageal or rectal, should be studied by a competent diagnostician or a group of coöperating specialists, and in all cases of adults with such symptoms a thorough examination should be made. It is our routine practice to do gall-bladder visualization tests, as first advocated by Graham,

in all examinations of adults where gastric symptoms are present.

We believe that in all cases where gall-bladder disease can be demonstrated, the gall-bladder should be removed, cystic duct drainage instituted and the ducts washed out.

CONCLUSIONS

1. Subserous cholecystectomy, in our opinion, is the operation of choice in chronic cholecystitis with or without stones.

2. Removal of the cystic duct is not necessary and adds to the hazard of the operation.

3. Cystic duct drainage will lower the mortality.

4. Subserous cholecystectomy with cystic duct drainage will reduce to a minimum operative accidents due to anomalies of the ducts and blood vessels.

5. This procedure also reduces to a minimum the danger of postoperative hemorrhage and bile peritonitis.

6. Cholangiography may be carried out routinely at the operating table without any appreciable loss of time, thereby eliminating the possibility of leaving undiagnosed stones or other obstruction in the common duct.

7. This procedure will obviate the necessity of exploring the common duct in many instances, thereby reducing the attendant hazards.

REFERENCES

1. Doyen, E. Surgical Therapeutics and Operative Technique. Vol. 3, pp. 125–7.
2. Sweek and Woodman. Cholecystectomy with cystic duct drainage as a routine procedure. *Am. J. Surg.*, 3: 573 (Dec.) 1927. Sweek and Patterson, A practical addition to the technic of cholecystectomy. *Southwest. Med.*, (Aug.) 1928.
3. Ochsner, A. J. Technique of gall-bladder operations. *Surg., Gynec. & Obst.*, 2: 204, 1906.
4. Reid and Halstead. Drainage of the common duct through the cystic duct; cysticocholedochostomy. *Ann. Surg.*, 78: 459, 1921.
5. Farr, R. M. Textbook on Local Anesthesia. Phila., 1929. Lea and Febiger.
6. Walters, W. Pathological physiology of stone in the common bile duct. *Surg., Gynec. & Obst.*, 63: 417, 1936.
7. Best, R. and Hicken, N. F. Cholangiographic demonstration of biliary dyssynergia and other obstructive lesions of the gall-bladder and bile ducts. *J. A. M. A.*, 107: 1615 (Nov. 14) 1936.
8. Holman, Emile. Technique of gall-bladder surgery. *Surg., Gynec. & Obst.*, 41: 778 (Dec.) 1925.

FOREIGN BODIES IN THE BLADDER

PAUL H. NITSCHKE, M.D.

PORTLAND, OREGON

THE vast majority of foreign bodies reach the bladder through the urethra, and therefore the size and shape of these objects are largely dictated by the caliber and curve of the urethra. In the female a short, comparatively straight tube allows these bodies to pass fairly readily to the bladder. In the male, however, a foreign body to reach the bladder must be able to adapt itself to the urethral curve, or come to rest in the bulbous portion. The floor of the urethra is quite elastic, and so inserted objects which hug the floor may so furrow it that they too come to rest in the bulb. Pathologic processes, such as urethral stricture and prostatic hypertrophy, may also cause arrest of the foreign body before it reaches the bladder.

Foreign bodies may be removed from the urethra by manipulation, by instrumentation through the urethroscope, or, if infection has made its appearance, by external urethrotomy in the perineal portion.

Foreign bodies may be introduced into the urethra for sexual stimulation either by the patient or his partner, with the blunt end introduced first. In some instances (Fig. 1) the patient may have tied a thin cord to the distal end of the object to facilitate removal. As the distal portion is the pointed end, a tug on the cord only serves to imbed the tip in the floor of the urethra.

An object may become lost in the urethra when the patient undertakes to treat himself, as was the case with the old gentleman who erroneously concluded that the cause of his urinary difficulty was just inside the external urinary meatus. He inserted a hair pin for theraputic purposes, which immediately disappeared from sight. This occurrence is more frequent in women, who in attempted abortions misdirect articles which find their way into the urethra and thence to the bladder. Finally, the physician may lose an article in the bladder during urethral instrumentation or cystoscopy, through breakage. These instances are usually of minor concern, for the operator is cognizant of the fact and removes the object at once or as soon thereafter as is practicable.

Migration of foreign bodies from adjacent structures to the bladder is not uncommon. Small missiles or a portion of an unremoved projectile may after a period of time wander from adjacent structures and finally come to rest in the bladder. Thus also a bone peg[1] or a pin,[2] inserted for fixation of the head of the femur, may find its way to the bladder. Rupture of a pathologic viscus, such as a dermoid cyst of an ovary, into the bladder has been reported. Gross[3] recorded the occurrence of a tooth, and Keyes[4] the presence of hair in the bladder from such occurrences.

Foreign bodies may also reach the bladder from the intestine. They may be swallowed and become the inciting agent of vesico-intestinal fistula formation, or occur secondarily as a sign of enterovesical fistula, in which case the foreign substance noticed may be undigested food material. In an instance of our own, fruit seeds were thus found. Though this condition is often associated with intestinal diverticulitis, in our series no diverticula were present.

In operations on the bladder and prostate, a foreign body may be left behind. This may consist of a portion of enucleated tissue or an article from the operator's armamentarium. With the present aid of trained staffs such accidents are indeed rare.

Even suture material may serve as a foreign body in the bladder. This can be obviated by catching all layers of the bladder except the mucous membrane in

closure. Many operators dispense with this precaution, for the use of absorbable sutures has made it largely unnecessary. We have seen, however, two instances in which the patient complained of urinary symptoms, with beginning calcareous incrustation around absorbable suture material. Both instances were cases in which the bladder had been "caught" during an extravesical operation. The material appeared to be chromic catgut. In another instance a surgeon unintentionally opened the bladder during a herniotomy. In closing the bladder the surgeon used linen sutures and included the mucous membrane in the closure. Stone formation resulted on each of the five sutures. (Fig. 4.)

As a general rule, infection and incrustation take place early. The salts deposited are usually phosphatic. The deposition takes place more rapidly on rough surfaces than on smooth ones, usually beginning near the center surface of the foreign body as this is the part more nearly at rest.

Wax also becomes incrusted. Young[5] reported as the first case of floating bladder calculus, an instance in which a coil of paraffin wax had become incrusted with urinary salts. In a case of our own (Fig. 5) some salt deposition was present on the foreign body, a variety of beeswax, in less than three weeks.

A foreign body tends to lie in the transverse axis of the bladder, due to the fact that the transverse diameter of the fixed bladder base is the greater. Should a well defined retrotrigonal bas fond be present, the foreign object will assume the transverse position in this depression. This fact severely limits the possibility of the object being passed without interference.

In the female, rigid foreign bodies of unusual length may be caught in a fold of the bladder wall immediately after introduction, and will therefore lie in a variation of the plane of introduction, or the anteroposterior diameter of the bladder. In the male, such articles would not negotiate the urethral curve, and so would not pass the bulb.

Objects of low specific gravity assume the transverse position only when the bladder is empty, for articles of gum or wax

Fig. 1. *X*-ray of nail in the bulbous urethra. A black thread, which had been tied to the distal end of the object to facilitate removal, protruded from the urethra. Pressure on the perineum caused a few drops of foul smelling bloody pus to exude from the external meatus. External urethrotomy was done. This showed several centimeters of the urethral floor to be necrotic due to associated infection. (Bellevue Hospital.)

float and thus rise to the uppermost portion of the bladder.

A foreign body may cause slight or no symptoms for some time, or, on the other hand, the symptoms of marked bladder irritation may be present almost immediately after introduction. The larger articles may cause a cessation of the urinary stream. With the advent of infection, which is usually introduced with the foreign body, symptoms of cystitis supervene. If the patient does not seek the advice of a physician through fear, sooner or later the distressing frequency and dysuria causes him to seek relief. These patients may also complain of some suprapubic and perineal pain which limits their activity. Hematuria is usually due to the accompanying cystitis, but in some instances it may be caused by the trauma of the foreign body.

Before any operative procedure is attempted, a careful history should be taken and the patient subjected to a physical

examination. For quite obvious reasons, examination of the perineum in the male should not be omitted. Rectal examination and bimanual examination should be done as a routine. Examination of the rectum and vagina should not be neglected, especially in children.

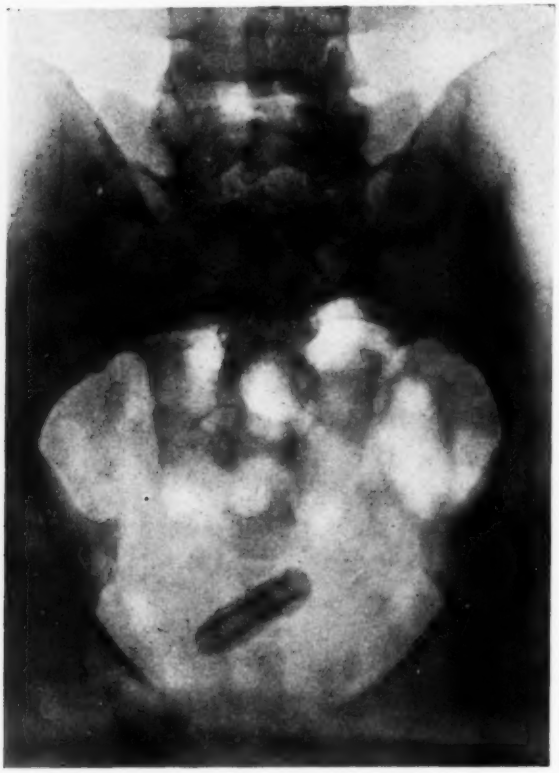


Fig. 2. Flat *x*-ray plate of an incrusted glass tube lying transversely in the bladder of a young adult male. The tube was inserted per urethra. It was removed by the suprapubic route. (Bellevue Hospital.)

A flat plate of the abdomen should be taken routinely. It not only serves to substantiate the patient's history but often obviates cystoscopy. This is well illustrated in Figure 2, where cystoscopic instrumentation of the incrusted glass tube would be dangerous, and in Figure 3 where the size of the stone and the danger of bladder perforation by transurethral manipulation make suprapubic cystotomy imperative.

As renal damage may occur through back pressure, associated infection, or coexisting pathology, renal function tests and blood chemical determinations should be carried out before any serious attempt is made to remove the foreign body.

Treatment consists in removal of the foreign body. The method of removal is dictated by the physical properties of the foreign body and the path by which the article reached the bladder. Small objects introduced per urethra may be removed cystoscopically with the aid of a cystoscopic forceps. Larger foreign bodies are often best removed by the use of one of several cystoscopic rongeurs designed for such purpose. Trauma to the urethra and bladder must be avoided in these manipulations.

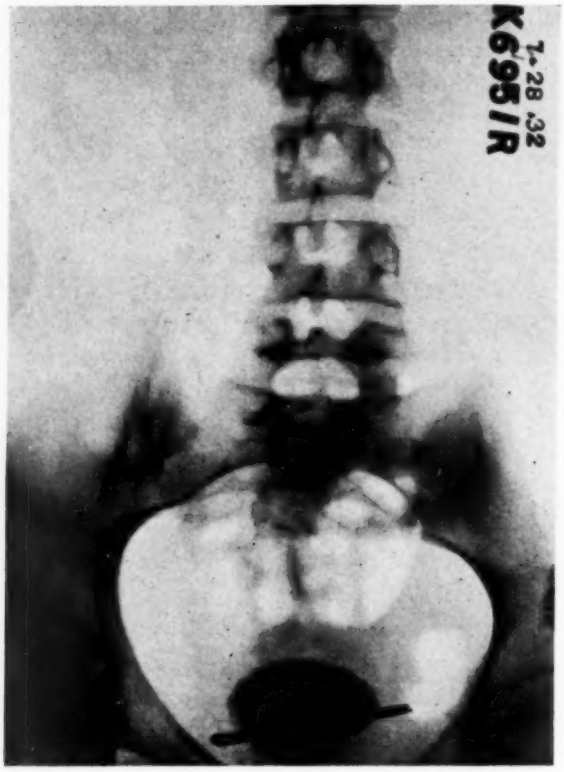


Fig. 3. Stone formation on a bobbie pin lying transversely in the bladder of a young girl. The incrustation evidently began near the center surface. The mass was palpable through the lower abdomen. It was removed by the suprapubic route. (Bellevue Hospital.)

Some articles present special difficulties. Thus Kimbrough[6] was unable to turn a hair pin in the bladder of a 4 year old girl, without running the risk of severe traumatization. By the use of an improvised snare

he was able to loop the free ends of the pin and draw these tightly to the scope. Articles of glass may be small enough to be easily removed through the cystoscope. Should there be any danger of shattering the glass object, then, even though small, it had best be removed suprapubically.

Pliable cord-like articles introduced into the bladder per urethra may be removed cystoscopically. If the article inserted is of some length it may have become so entwisted or knotted that removal through the urethra is impossible. Removal by cystotomy is then the only recourse.

With few exceptions foreign bodies which have migrated from adjacent structures to the bladder are best removed suprapubically. However, if the object is small and has not given rise to permanent sinus or fistula formation it may be removed per urethra by cystoscopic instrumentation. Crane and Moody[7] using an F. 18 panendoscope, removed a splinter of wood from the bladder of a 7½ year old boy one month after accident. A smaller piece had previously been removed from the urethra where it had become impacted. In this instance, the boy fell astride a picket fence and the splinter was driven up the inner aspect of the thigh through the obturator foramen into the bladder.

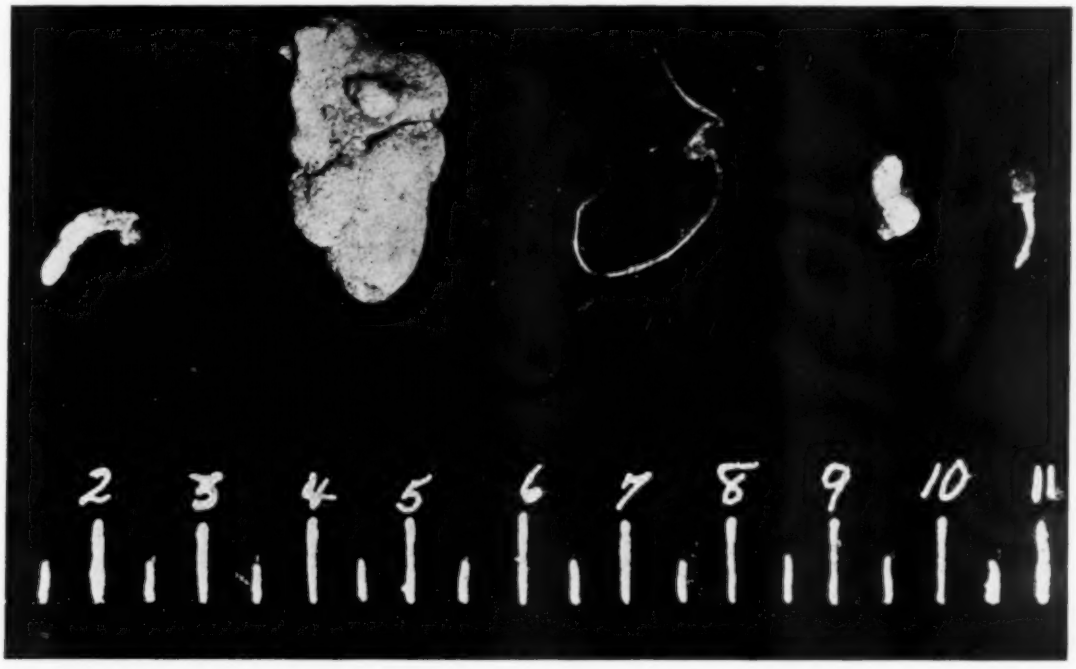


Fig. 4. Stone formation on five non-absorbable linen sutures used in bladder closure. Incrustation removed from one suture. Removal by instrumentation was impossible because of position and danger of bladder perforation. Sutures were removed by suprapubic cystotomy. (Scale in centimeters.)

Bone pegs and pins, used for fixation of the head of the femur, which have migrated to the bladder are best removed by suprapubic cystotomy. The curettement or excision of a sinus tract may be essential, and after closure of the bladder defect, extravesical drainage to facilitate healing is often necessary.

Where the foreign body is due to associated pathology of an adjoining organ, such as a dermoid cyst of an ovary, surgical removal of the pathologic process is indicated. After closure of the bladder sinus suprapubic drainage by means of a Pezzer catheter is safer than catheter drainage per urethra.

The diagnosis of vesico-intestinal fistula is not difficult. The symptoms of cystitis, such as frequency and dysuria with pyuria, pneumaturia, and the passage of fecal matter and undigested food particles, is

quite conclusive. Whether the fistula is the result of the foreign body or the foreign particles the result of the fistula is not so important. The primary concern is the eradication of the fistulous tract. The presence of malignancy or of a specific inflammatory process should be ruled out. Before operation an effort should be made to determine the exact level of the portion of the bowel involved. This point may definitely influence the operative procedure. For instance a colostomy would be of no value if the fistula involved the small bowel.

Fig. 5. Beeswax removed from the bladder of a 16 year old boy. As beeswax is brittle, several sharp blows with a closed snare, improvised from a double strand of piano wire and a ureteral catheter, sufficed to break the foreign body into several pieces, which the patient passed on voiding. Two portions not shown were lost down the drain. (Scale in centimeters.)

Cystoscopy will locate the exact position of the fistulous opening in the bladder. Catheterization of the vesical stoma of the fistula and injection of an opaque solution may be helpful or cystograms may be made with an attempt at backflow of the opaque solution into the intestine. Severe reaction following the use of sodium iodide in the latter procedure has, however, been reported.[8] Fluoroscopy and *x*-ray of the intestinal tract after the administration of barium by mouth or enema as indicated is most important. In fistulae involving the distal portion of the large bowel, sigmoidoscopic and proctoscopic examination may be indicated.

As a rule, during operation for closure of an enterovesical fistula, the intestine is first disposed of, for should it become necessary to terminate the operation at any time, it can be done thereafter with a minimum amount of risk to the patient. The operative procedure may involve only the removal of the appendix, simple closure of the intestinal opening or a bowel resection. In the case reported by Herbst and Miller,[9] where the fistula was caused by a bone from the intestinal tract, three separate operations with several weeks interim were necessary. The first stage consisted of freeing the appendix from the cecum, and resecting a portion of the involved sigmoid. This mass was left adherent to the bladder. A colostomy was done to sidetrack the fecal stream. The second operation involved the removal of the resected mass from the bladder and closure of the vesical defect. The third stage consisted of a lateral anastomosis to establish continuity of the large bowel.

In our own case of fistula involving the bladder and sigmoid portion of the large

bowel, the fistula recurred after the first operation. Bladder drainage had been accomplished through an in-dwelling catheter. Prior to the second operation a colostomy was done. After closure of the fistula had been effected the bladder was drained suprapubically through a large Pezzer catheter. Recovery was uneventful. The patient has been well for over two years.

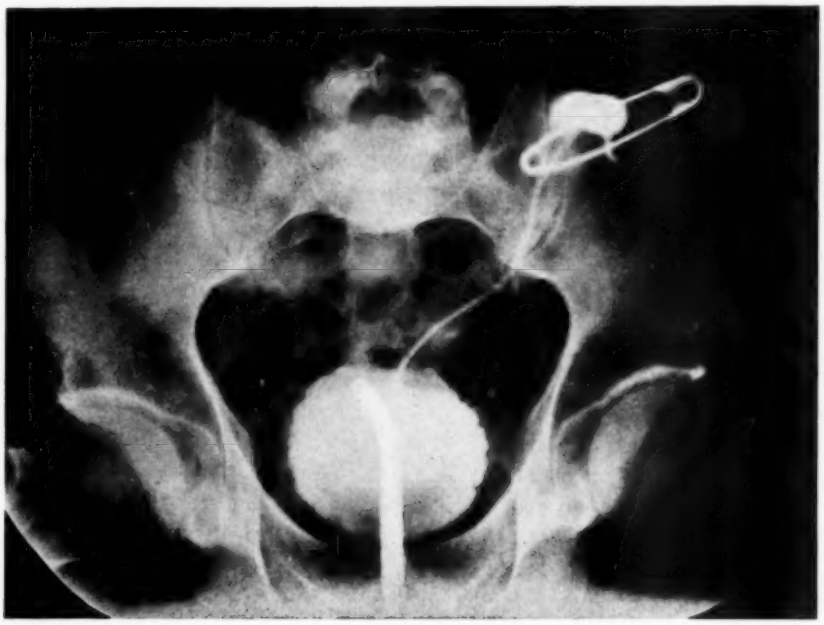


Fig. 6. Ureteral catheter passed into the sigmoid colon from the bladder, through intestinovesical fistula. Note the relationship of the colostomy performed to divert the fecal stream and relieve tension prior to operation for closure of the fistula. The opaque solution injected to outline the tract has run back into the bladder.

Because of their physical properties, foreign bodies of gum or wax should be considered separately. Paraffin wax may produce a cystitis within twenty-four hours. Though wax and gum objects are usually introduced in pencil form, they tend to curl up due to body warmth and the contractile force of the bladder. This makes passage on voiding impossible. If the wax pencil introduced is comparatively thick, or the object is a wick-containing candle then the curling up process is delayed. Such bodies, like rigid objects, will come to rest in the transverse diameter of the bladder base as the bladder empties. The slender wax pencils curl up quite readily. In the beginning of this process the coils usually maintain their identity and a composite mass does not form until later, especially in the case of the firmer waxes. The importance of this observation is shown in the example of a lad of 16, who for the purpose of sexual excitement, introduced into his urethra an improvised pencil of a variety of beeswax (Fig. 5) and promptly lost it. The ensuing cystitis caused him to seek relief. While trying to remove the irregularly coiled wax body from the bladder by means of a snare, we noted that an elbow of the coiled wax broke off when struck with the closed snare. As this particular type of wax is quite brittle, several sharp blows broke the object into a number of small pieces, after which the cystoscope was withdrawn. The patient voided the particles before leaving the office.

In another instance, because of the buoyancy of the wax object in a water medium and poor visibility in that portion of the bladder directly above the bladder neck, the bladder was filled with mineral oil and the piece of wax removed from the

floor by means of a Young's rongeur. One should guard against overdistention of the bladder in this procedure. O'Neil[10] described this method in 1931, using an olive oil medium, but recommended the use of mineral oil as olive oil becomes rancid. In the female the use of the open air cystoscope makes this procedure unnecessary.

Gum rapidly forms a composite mass in the bladder. In two cases we have removed portions of gum from the bladder by means of a Young's rongeur, but in both a portion of the gum remained in the bladder. In each instance the remaining gum was passed by the patient shortly after instrumentation. Gum, like wax, also sinks in mineral oil.

Although we have never used solvents to remove gum or wax from the bladder, there are instances where the solvent method is superior to any other. However, if encrustation has taken place on the foreign body, the action of a solvent is nullified and recourse must be had to some other procedure.

The removal of gum or wax from the bladder by means of solvents may be accomplished by any one of three methods. The undiluted solvent may be used, being injected into the bladder through a catheter. Lyons[11] reported an instance where the patient was allowed to void the bladder contents after benzene had been injected for the removal of chewing gum. This resulted in a severe urethritis, with some blistering and infection of the scrotum and thighs where the fluid had dribbled.

Harris[12] and later Katzen[13] injected warm mineral oil into the bladder to rid the patient of a wax candle. The patient retained the oil several hours. Additional measures such as gentle massage over the bladder region and heat by means of hot water bottles and hot sitz baths were used.

Caples[14] and later Melin[15] removed a wax pencil from the bladder by using a mixture of one third low grade gasoline (55° Baume scale) and paraffin oil; 125 c.c. of this mixture was injected after it had been heated to 110° F. This was retained for several hours by the patient. A number of daily treatments were necessary.

In the method used by Hattinger[16] the bladder is filled with boric acid solution or sterile water and then 12 c.c. of xylol is run in through the catheter. As the xylol floats on the surface of the water, much of the bladder mucosa is unexposed to its effect. By having the patient change his position from time to time, exposure of any one portion of the bladder to the concentrated solvent over a lengthy period is avoided. In Hattinger's case the patient passed a milky solution containing the dissolved foreign body after two hours. Cystoscopy thereafter was negative. This procedure was later used by Turner[17] with equal success, the solvent in this instance being withdrawn through a catheter.

Prior to instrumentation or the use of solvents it may be advisable to irrigate the bladder for several days to alleviate the distress due to the accompanying cystitis. All solvents used should be run into the bladder through a catheter. After the allotted time has elapsed the bladder should be emptied by means of a catheter and the bladder irrigated to wash out any remaining solvent.

The use of solvents, such as xylol, carbon tetrachloride, gasoline or kerosene, in large quantity should be avoided. Their use is not without some danger. Ether[18] because of its escharotic action should never be used. Mineral oil can be used in quantity and safely applied over a long period of time. Though its action is comparatively slow (Fig. 7), the fact that the wax body need be partially only dissolved in order to be passed makes mineral oil an ideal solvent. However as a solvent for the firmer waxes such as beeswax, and for chewing gums, mineral oil is comparatively useless. (Table 1.) Gum or beeswax may be removed by instrumentation as previously described, or a solvent such as xylol or benzene may be employed according to the method of Hattinger. It is interesting to note that gum is usually chewed before it is inserted into the urethra. This factor

makes cystitis almost a certainty but aids the action of the solvent, for gum exposed to a solvent without first having been chewed or placed in a watery solution, dissolves very slowly.

When the foreign body has been removed, whether by instrumentation or solution, the bladder should be examined several days later to ascertain that no foreign particles remain. In some instances, cystoscopic instrumentation is impossible, or if the article is of a variety which makes instrumentation hazardous, removal by suprapubic cystotomy is indicated. The obliteration of the channel formed by the object in its migration to the bladder may necessitate additional surgery.

Foreign bodies of gum or wax may be removed cystoscopically. Waxes with comparatively high melting points, such as

TABLE I

COMPARATIVE SOLUBILITY

Commercial Wax	12 C.c. Xylol	12 C.c. Benzene	12 C.c. Carbon Tetrachloride	12 C.c. Gasoline	12 C.c. Kerosine	25 C.c. Mineral Oil	25 C.c. 1/3 Gasoline 2/3 Mineral Oil
Paraffin	55 min.	1 hour.	55 min.	1 hour 10 min.	3 hours.	9 hours.	2 hours.
Crayon	55 min.	1 hour.	55 min.	1 hour.	2¾ hours.	10 hours.	2½ hours.
Candle	45 min.	1 hour.	1 hour 10 min.	1 hour 20 min.	2¼ hours.	8½ hours.	2½ hours.
Beeswax	4 hours.	5 hours.	4 hours.	8 hours.	32 hours.	Slight action in 72 hours.	Some action in 72 hours.
Chewed chewing gum	30 min.	30 min.	30 min.	30 min.	2 hours.	Slight action in 72 hours.	Slight action in 48 hours.

Articles used are those which have been reported as vesical foreign bodies. Specimens were 1 inch long by ¼ inch in diameter. One inch of common chewing gum stick was chewed for fifteen minutes. The temperature was kept at 37.5°C. and the specimens were shaken every fifteen minutes for ten hours. Note the relative insolubility of beeswax and chewing gum in mineral oil and one-third gasoline and two-thirds mineral oil. Note also the slow action of kerosene on beeswax.

as with a candle, the insoluble material, in this instance the wick, may require removal through a cystoscope.

SUMMARY

Most foreign bodies reach the bladder per urethra. They may also migrate from adjacent structures or from the intestine, in which case the presence of an enterovesical fistula may further complicate the picture.

The mode of removal of foreign articles is largely dictated by their physical properties and the path by which they reached the bladder. In many instances, removal of these foreign bodies may be safely accomplished by cystoscopic instrumentation. If beeswax, are brittle and can be broken up into smaller particles through the cystoscope, after which they will be passed with little difficulty. Wax and gum may also be disposed of through the proper use of solvents. The use of concentrated solvents in large quantity should be avoided. Mineral oil is an exception to this rule. If concentrated solvents are used they should be employed in small quantities and in such a manner that the bladder trigone is not exposed.

I wish to thank my former chief and teacher, Dr. A. R. Stevens, Director of the Department of Urology, Bellevue Hospital, New York City, for his permission to use Figures 1, 2, and 3 in this article.

REFERENCES

1. Grant, Owsley. An unusual foreign body (bone peg) in the bladder. *J. A. M. A.*, 107: 1632 (Nov. 14) 1936.
2. Ravenswaay, Alex. van. Steel pin inserted into neck of femur found in bladder. *Am. J. Surg.*, 31: 566 (March) 1936.
3. Gross, Samuel D. System of Surgery. Ed. 6. Phila., 1882. Henry C. Lea. Vol. 2.
4. Keyes, Edward L. Urology. New York, 1928. Appleton.
5. Young, H. H. Practice of Urology. Phila. 1926. W. B. Saunders. Vol. 2.
6. Kimbrough, O. T. Removal of hairpin from the bladder of a four-year-old child with an improvised instrument. *J. Urol.*, 28: 251, 1932.
7. Crane, J. J., and Moody, E. E. A splinter of wood lodged in the urinary bladder. *J. A. M. A.*, 104: 1702 (May 11) 1935.
8. McCarthy, Joseph F. Discussion of article by Fowler, H. A. Intestinovesical fistula. *Tr. Am. A. Genito-Urinary Surgeons*, 22: 371, 1929.
9. Herbst, R. H., and Miller, E. M. Vesico-intestinal fistulas caused by foreign bodies in the bowel. *J. A. M. A.*, 106: 2125, 1936.
10. O'Neil, Richard F. Unusual foreign body in the bladder. *Tr. New England Branch of Am. Urol. A.* p. 24, 1930–1931.
11. Lyons, Oliver. Discussion of article by Morris, H. L., and Owen, C. I. Chemical solvents used in dissolving foreign substances in the urinary bladder. *J. A. M. A.*, 81: 1671 (Nov. 17) 1923.
12. Harris, Augustus. A case of paraffin foreign body in the bladder. *Med. J. & Rec.*, 134: 372 (Oct. 21) 1931.
13. Katzen, Perry. Paraffin foreign body in the bladder. *J. A. M. A.*, 105: 1422, 1935.
14. Caples, B. H. Foreign body in the urinary bladder. *Surg., Gynec. & Obst.*, 29: 315, 1919.
15. Melin, David R. Nonsurgical removal of paraffin in the urinary bladder. *J. A. M. A.*, 80: 685 (March 10) 1923.
16. Hattinger, R. Cited by Harris, Augustus.[12]
17. Turner, J. Harolde. A foreign body of paraffin in the urinary bladder removed with xylol as solvent. *J. Urol.*, 33: 471, 1935.
18. Morris, Harold L., and Owens, Clarence I. Chemical solvents used in dissolving foreign substances in the urinary bladder. *J. A. M. A.*, 81: 1667, 1923.

Vaginal polyps are occasionally found in children. Their discovery usually follows upon the investigation of vaginal bleeding.
From—"Pediatric Surgery" by Edward C. Brenner (Lea & Febiger).

THE GYNECOLOGIC PATIENT WITH URINARY SYMPTOMS*

SAMUEL HOCHMAN, M.D.

Senior Clinical Assistant, Mt. Sinai Hospital; Assistant Visiting Gynecologist, Harlem Hospital

NEW YORK CITY

THIS analysis of 200 consecutive female patients with urinary symptoms is from the cystoscopy clinic of the Gynecologic Division of the Mt. Sinai Hospital, New York City.

Where gross urologic conditions such as tuberculosis, neoplasms, calculi, etc., were found in the course of our gynecologic urologic investigations, the patients were transferred to the urologic service. Such lesions as urethritis, submucous infiltrations, inflamed glands, stricture and neoplasms of the urethra were ruled out by routine endoscopy, or when found received appropriate treatment. These are not included in the cases reported here.

After excluding the above cases, there are left a host of minor lesions and bladder disturbances, which, however, play a very major rôle in the discomfort and unhappiness complained of by gynecologic patients. They challenge all of our resources in their treatment. It is only by the most meticulous accuracy in localization, the utmost gentleness and niceness in manipulative procedures, the utilization of a variety of physical, hydrotherapeutic, chemical and psychotherapeutic measures, and the exercise of extraordinary patience that we are able to obtain adequate results. A special cystoscopic clinic therefore is of inestimable value, and constitutes an essential part of a gynecologic out-patient department.

This analysis is discussed under the following headings: age, marital status, obstetrical and surgical trauma, menopause, symptomatology, pathology and treatment. The purpose of this study was to help clarify the correlation of symptoms to definite bladder disease, the applicability of specific treatment for specific pathologic conditions and to standardize our treatments based on cystoscopic findings.

Age. The average age in this series is 44.5 years; 70, or 35 per cent, were in the menopause, and of the latter, over a third complained of typical menopausal symptoms. At the menopause, among the recognized factors in the genesis of urinary symptoms are the local atrophy, the increased sensitivity to moistening by urine, and the lowered threshold of the vasomotor and sympathetic systems. Urinary symptoms in this group, part of the larger picture of the menopause, react well to general sedation and, in selected cases, to hormonal therapy.

Marital Status. The greater proportion, 97 per cent, were married or had been married. The effect of marital maladjustment as related to various bladder symptoms, and particularly the rôle of coitus interruptus, properly belong to the field of scyphotherapy. The cases belonging to this group have been separately studied by Dr. Max Mayer in his special clinic, and will not be included in this report. It is well recognized that psychogenic factors may play a considerable part in urinary symptomatology. In this series, an amazing number (50, or 25 per cent) were found to have had some form of neurosis. This number includes those with menopausal symptoms accompanied by mental disturbances of varying degree. Many of the patients who complained considerably of urinary symptoms, were found to have a normal bladder or a mild trigonitis, and

* From the Gynecologic Out-Patient Department of the Mt. Sinai Hospital, New York City.

were not relieved by local treatment unless accompanied by some form of psychotherapy.

Gravidity. Seventy-nine, or 90 per cent, of the patients were parous. Gravidity is of importance in the production of bladder symptoms because of the not infrequent cystoceles, urethroceles, prolapses, etc., as well as the low grade infections of lacerated cervices. The latter should not be neglected in the treatment of recurrent bladder infections. At this point we may call attention to the importance of treating vaginitis, especially trichomonas vaginalis vaginitis, with or without parasitic invasion of the urethra. Seventy-five, or 38 per cent, showed evidences of obstetrical trauma; 53 per cent had cystoceles, 12 per cent urethroceles and 22 per cent lacerated cervices of the remainder, many showed cystorectoceles, prolapses, etc.

Surgical Trauma. Ninety or 45 per cent of the patients had had some operative procedure. Of these, 27 per cent had had plastic repairs of birth injuries and 30 per cent other gynecologic operations. It seems probable that the mobilization of the bladder from above or below occasionally interferes with its innervation or vascular supply and predisposes to local symptoms. Ordinarily, the postoperative cystoscopic examinations following anterior colporrhaphy show a surprising degree of restitution to normal. In some instances there is seen, for a time, a median ridge formation with increased vascularization of the mucosa and a tendency to bleeding on the slightest manipulation. Rarely, a penetrating stitch causes a temporary superficial ulceration or localized bullous edema. Excessive plication may cause temporary dysuria and even retention, but these symptoms respond with great rapidity to the simplest measures, such as periodic emptying of the bladder and occasional bland irrigations. Occasionally a distinct bar formation is seen instead of a median ridge. This group seems to respond readily to the passage of a sound. The ureters were never found compromised as a result of anterior wall plastic repair or parametrial fixation operations.

Anatomy and Pathology. Since this special clinic also served the function of instruction in cystoscopy and gynecologic urology to the younger members of the staff, we have emphasized the importance of a systematic search for any urologic lesions. In the instruction, the importance of diagnosing the exact site of the inflammation, congestion, or edema has been emphasized, for such a differentiation is vital in the choice of therapy. For purposes of cystoscopic orientation, the urinary tract was divided into the following divisions with the common lesions to be sought under each of the headings:

I. Urethra:
   (1) Caruncle
   (2) Urethrocele
   (3) Relative stricture
   (4) Urethritis
II. Internal sphincter:
   (1) Excursion disturbances
   (2) Edema
      (a) Villus formation or papillomatous excrescenses
   (3) Bar formation
   (4) Congestion
III. Bladder neck or vesico-urethral junction:
   (1) Edema (bullous and cystitis cystica)
   (2) Congestion
   (3) Exudate
IV. Trigone:
   (1) Edema
   (2) Congestion or hyperemia
   (3) Exudate
   (4) Distortion
      (a) Pressure by fibroid, etc.
V. Fundus and rest of bladder:
   (1) Distortion
   (2) Diverticulum
   (3) Ulcer
VI. Ureteral orifices:
   (1) Appearance (edema or exudate)
   (2) Location
   (3) Number (accessory)

VII. Ureters and kidneys:
(1) Stricture
(2) Dilatation
(3) Calculus, etc.

The internal sphincter is treated here as a special entity, separate from the bladder neck with which it usually has been included. Normally when the cystoscope is turned laterally, the sphincter resembles a crescent or quarter moon, and its lower or posterior portion does not project above the surface and is not sharply differentiated, but seems continuous with the posterior urethra and floor of the bladder. However, where edematous, it may loom up in a bar formation as a barrier beyond which it is difficult to see the bladder neck. Sometimes the entire trigone or the margin may become irregular, with a scalloped appearance or lined with polypoid excrescenses.

The area between the internal sphincter and the trigone may be called bladder neck or vesico-urethral junction. This is often the site of a simple or a bullous edema. Carcinoma of the cervix or uterus and pyosalpinx, which may be the source of a bullous edema, must be ruled out. An exudate may cover this area and extend over the trigone. Here the pathology may vary from a simple hyperemia, which is most common, to a marked edema with obliteration of the blood vessels. Narrowing of the urethra or relative stricture should always be looked for, as it is often the source of severe bladder symptoms, even though an ordinary metal catheter may be passed easily.

Lesions of the upper urinary tract are excluded by ureteral catheterization and intravenous and retrograde pyelography. When pathologic conditions are found, they are referred to the surgical division for treatment. The conditions found are listed in Table 1 (description involves considerable overlapping).

One hundred and forty-nine patients, or 75 per cent, had lesions at or near the bladder neck. Including bar formation and relative stricture, fifty-four, or 27 per cent, had edema of the internal sphincter. It is

Table 1

	No. of Cases	Per Cent
Edema of bladder neck	77	38.5
Edema of internal sphincter	30	15
Bar formation	24	12
Congestion of the trigone	38	19
Edema of the trigone	35	16.5
Edema of bladder neck and trigone	22	11
Relative stricture of urethra	18	9
Urethral caruncle	11	5.5

obvious, therefore, that the term cystitis usually applied to so many female patients with urinary symptoms is too broad. A generalized cystitis was rarely seen, as the bladder above and lateral to the trigone is usually normal.

At this point it may be well to consider the importance of the examination of the urine. A catheterized specimen is obtained from each patient when first seen. If the specimens contain frank pus or blood, ureteral catheterization is done as soon as feasible. With the exception of some instances of acute cystitis or upper urinary involvement, most of our cases showed a practically normal urine. A routine urine examination therefore cannot be relied upon as an index of a pathologic condition in the genitourinary tract, particularly in the bladder.

Symptomatology. The cardinal symptoms were in their order of importance, frequency, burning, incontinence, and urgency, occuring alone or in combination. One hundred and eight, or 54 per cent of the cases, complained of frequency of urination. Of these, twenty-nine did not return for treatment after cystoscopy. Of the remaining eighty-one, forty-six, or over 50 per cent, were cured or much improved and thirty-two somewhat improved.

Eighty-two, or 41 per cent, complained of burning urination. Urgency was recorded in only nineteen cases, but I believe it to have been more frequent than these figures indicated. Some of our standard terms, in a history, offer considerable difficulty in

their translation for foreign patients. It is also difficult to evaluate treatments for specific urinary symptoms, as they usually do not occur singly.

Incontinence was found in forty-five, or about 22 per cent of the cases. Edema of the sphincter and bar formation were more prominent in patients with this symptom. However, many of these cases also had frequency, burning and urgency. Of the forty-five, six were not cystoscoped and five were not treated, leaving thirty-four, of whom seven were cured, twelve much improved, ten somewhat improved and five not improved. An important part of the examination of each case of incontinence was a complete neurologic investigation to eliminate any possible lesion of the central nervous system.

Other symptoms, such as hematuria, pyuria, backache, pain in the loin and flanks, were investigated and treated in the usual manner. Nephroptosis is an occasional etiologic factor in symptom production.

We have assiduously avoided a blanket diagnosis of "cystitis," which in the past, has resulted in the routine irrigation and instillation therapy. Instead, by careful topographic differentiation, we feel that we obtain results that are quicker and more lasting.

Treatment. The treatment may be divided into general and local. In the former are measures used when the symptoms form part of a constitutional condition, such as neurosis, menopause, cardiac condition, etc. Local procedures consist of hot douches and hot sitz baths, bladder irrigations and instillations, bladder distentions, Kelly treatments, dilatations of the urethra with massage, or a combination of any of the above. Hot douches and sitz baths were used alone or with sedatives in cases of mild congestion of the trigone, bladder neck or of the rest of the mucosa. Symptoms very often improve without further treatment, particularly where associated with an inflammatory condition in the pelvis, such as salpingitis or parametritis. Of the thirty-eight cases of congestion of the trigone, thirty-two received this treatment alone or continued with irrigations. All patients were instructed to take prolonged hot douches and sitz baths daily in conjunction with any of the other treatments for the general effect produced by heat and hydrotherapy. When indicated, diathermy was used freely as a physiotherapeutic adjuvant.

Bladder irrigations with or without instillation of argyrol, silver nitrate, etc., were the treatments given for a more severe trigonitis, cystitis or considerable injection of the bladder neck.

Hydraulic distention of the bladder is used in a limited number of cases where the capacity of the bladder is diminished, due either to chronic cystitis or to extravesical pressure. These cases have frequency as a major symptom, and when they are enabled to hold at least 300 c.c., their symptoms are greatly improved.

Heretofore in this clinic, most of the cases of edema of the sphincter, vesicourethral junction and trigone were routinely given topical application of 5 per cent silver nitrate over this area, as described by Kelly. In an analysis, it was found that a great many returned to the clinic repeatedly with little or no improvement. This treatment, often in conjunction with dilatation, was used for edema of the bladder neck, especially bullous edema and severe edema of the trigone, particularly where an exudate covered this area. However, as it was found that many of these cases were completely relieved by dilatation of the urethra, this is being used more frequently at present.

Dilatation, or the introduction of Hegar dilators or sounds past the internal sphincter, accompanied by massage of the urethra on the sound by the vaginal finger is a form of treatment used frequently in this clinic. The technique and results of this treatment, particularly for incontinence and enuresis had been noted as far back as 1888, when Oberlander described incontinence as an irritation of the reflex caused by the

urethral muscles in the region of the sphincter, and recommended urethral dilatations. Since then, Sanger and Credé in 1890, Bagot in 1891, and others up to Hinman in 1935, have reported success with this method. It was a theory of most of these writers that incontinence was caused by a weakness of the internal sphincter. The beneficial effect of the procedure depended on the fact that the sphincter, through the irritation of the stretching, would reflexly contract more strongly, increase muscle tone and set up a certain degree of work hypertrophy.

Treatments are given weekly, gradually increasing the caliber of the dilators, until a No. 10 or 11 Hegar or a 28 French sound is reached. Usually, not more than six to nine treatments are given, and often the symptoms subside following one treatment. This may throw light on the occasional cessation of symptoms following a simple cystoscopy. This treatment was found to be almost specific for edema of the internal sphincter or bar formation and relative stricture of the urethra. In the latter cases, although the urethra admitted a catheter easily, results were not obtained until it was possible to introduce painlessly a sound of a larger size. Table II shows the results obtained in those cases treated by dilatation.

Table II

	No. Dilated	Cured or Greatly Improved	Improved	Not Improved	No Check-Up
Edema of the bladder neck	44	30	7	1	6
Edema of sphincter	27	18	3	2	4
Bar formation	20	10	4	2	3
Relative stricture of urethra	18	12	4	2	0

Other methods of treatments were successful, but none so rapidly or permanently as the dilatation. We have, of course, not ignored the probability of a strong suggestive element in the treatment. Results of this treatment in the cases of incontinence were excellent when there was an edema of the sphincter and bladder neck. (Table III.)

Table III

	No. Dilated	Cured or Greatly Improved	Improved	Not Improved	No Check-Up
Incontinence	23	14	5	2	2
Frequency	37	25	7	2	3
Burning	37	23	10	3	1
Urgency	11	8	2	1	1

The eleven cases of urethral caruncle were cauterized. Eight were cured and three were only slightly improved, the latter having been found to have bladder disease.

SUMMARY

1. Our experiences with 200 consecutive minor urologic cases in gynecologic patients is here reported.

2. A special cystoscopic clinic is an essential part of a gynecologic out-patient department.

3. Topographic subdivision of the bladder is of value in practical diagnosis.

4. Treatment should not be routine, but should be specific for each type of bladder disease.

5. There are various constitutional and mental factors that are important in the selection of proper treatment.

6. In this series, over 90 per cent of the patients presented pathologic conditions about the bladder neck and internal sphincter.

7. Dilatation of the urethra is found to be of great value in this type of patient.

I wish to express my thanks to Dr. J. J. Squire for his help in the preparation of the tables, and to Dr. Max Mayer for his advice in the elaboration of this paper.

REFERENCES

1. Gasparjan, A. and Efstein, J. *Urologia*, 9: 8, 1932.
2. Oberlaender, M. *Berlin. klin. Wchnschr.*, 1888.
3. Jamin, R. *Ann. mal. org. Genitourin.*, 7: 507, 1889.
4. Oberlaender, M. Étiologie et traitement de l'incontinence d'urine. *Rev. mal. de l'enf.*, 1889.

5. Saenger, M. *Arcb. f. Gynäk.*, 38: 324, 1890.
6. Bagot, W. S. *Dublin J. Med. Sc.*, 42: 284, 1891.
7. Zuckerkandl, O. Handbuch der Urologie, 1: 689, 1904.
8. Frankl Hochwarth, Lother von. Handbuch der Urologie, 2: 777, 1905.
9. Bazy, M. P., and Deschamps, M. *Rev. de therap. med.-chir.*, 75: 724; 757, 1908.
10. Tuteur, Margaretha. Inaug.-Dissertation, Freiberg, 1927.
11. Dietel, Friedrich. Das Bett naessen. Seine Ursachen und Vorschlaege zu seiner Behandlung. Munich, 1929. J. F. Lehmann.
12. Stoeckel, W. Lehrbuch der Gynaekologie. Leipzig, 1931. S. Herzel.
13. Casper, Leopold. Lehrbuch der Urologie. Vienna-Berlin, 1932. Urban and Schwarzenberg.
14. Hinman, Frank. The Principles and Practice of Urology. Phila., 1935. W. B. Saunders.
15. Eberbach, Carl. W. in Davis' Gynecology and Obstetrics. Vol. 3, 1934.

Although no part of the body is immune [in children], certain regions are especially subject to the development of new growths. The kidneys and adrenals are most frequently involved and furnish approximately 30 per cent of the malignancies of childhood. The bones, brain and meninges, and the eye and orbit are next most common sites and each accounts for approximately 10 per cent.

From—"Pediatric Surgery" by Edward C. Brenner (Lea & Febiger).

NON-SPECIFIC SALPINGITIS

RUDOLPH MARX, M.D., F.A.C.S.

Attending Staff, Cedars of Lebanon, Hollywood and Queen of the Angels Hospitals

LOS ANGELES, CALIFORNIA

THE experiences of gynecologists during the last two decades have brought out increasing evidence that there is no clinical and pathologic picture which is absolutely typical for a gonorrheal infection of the female pelvic organs in general and the Fallopian tubes in particular. Various other infective agents have been found capable of producing pictures resembling textbook descriptions thought to be characteristic of specific infection of the female pelvic organs.

GENERAL PATHOLOGY OF SALPINGITIS

The manner in which an infection enters the Fallopian tubes varies with the type and the virulence of the infecting organism. It may spread from the cervix through the uterine cavity, entering the tube through its uterine opening; it may enter from the peritoneal cavity through the ampulla, or it may be carried by the lymphatics or the blood stream either from neighboring organs or from some distant focus. When the infection enters through the uterus, both tubes are usually affected simultaneously.

In the acute type of inflammation, the tube is swollen, its tortuosity increased, the mucous membrane congested, and the fimbria reddened and swollen. There is a hypersecretion of mucus. Microscopically, the epithelium of the mucous membrane is swollen and has lost its cilia. The submucosa is infiltrated with leucocytes and round cells.

Depending upon the origin and type of infection, either the inflammatory process starts at the endosalpinx and spreads through the muscles to the peritoneal covering; or, if the infection has been carried through the lymph or blood vessels, the initial lesion is in the wall of the tube, spreading from there.

Unquestionably a large number of cases of salpingitis are abortive and do not go beyond the initial stage characterized by swelling, congestion, and mucous secretion of the tube. Even in this stage the adjoining structures and the ovary are usually found to participate in the congestion and edema, and some serous exudation shows an associated peritoneal irritation.

If the process does not advance beyond this stage of catarrhal salpingitis, complete resolution and restitution to the normal condition is usual. But when the inflammation progresses, the secretion of the tube becomes purulent, and inflammatory adhesions form, involving the fimbria, the inner folds of the mucosa, and the adjoining structures and ovary in varying degrees. Partial or complete obstruction of the tube is the well known sequela. Salpingitis nodosa is the final stage of a localized infiltration within the tubal wall. Hydrosalpinx, pyosalpinx and tubo-ovarian abscesses are common results of repeated attacks.

ETIOLOGY

As mentioned before, it is often difficult to differentiate between the evident clinical manifestations of non-specific and specific infections. Robert Cruikshank analyzed a recent series of 200 women sent to a venereal clinic as cases of clinical gonorrhea. He found that an exhaustive bacteriologic and serologic study failed to show evidence of gonorrheal infection in 45 per cent.

Statistics on the relative frequency of the various types of infections responsible for salpingitis differ greatly. A notoriously unreliable source of information as to the etiology of salpingitis is the personal history. In a total number of 545 cases of

salpingitis, Farr and Findlay obtained the history of gonorrhea in less than 3 per cent of the cases.

The main difficulty in discovering the etiology of salpingitis arises from the fact that the majority of tubes surgically removed have been found to be sterile.

A summary of statistics covering 753 cases from a number of European clinics, dating from a time when many tubes were surgically removed, showed: approximately 60 per cent sterile; 24 per cent containing gonococci; 11 per cent staphylococci and streptococci; 3 per cent tubercle bacilli; and about 1 per cent each of pneumococci and colon bacteria.

In a clinical, pathologic, and bacteriologic study of nearly 300 salpingitis cases, Curtis also found that the majority of tubes removed at operation were sterile. However, from the total evidence collected, he came to the conclusion that more than 70 per cent of the lesions were most likely produced by a gonorrheal infection. Five per cent were of tubercular origin and more than 15 per cent were entirely due to other pus producing bacteria, notably various types of streptococci.

It is a well known fact that gonococci survive for only a short time in pus tubes; and if not reinfected from the cervical glands, in which the gonococci can live indefinitely, pus tubes become sterile within a few weeks. On account of this experience, the cases of salpingitis found to be sterile were commonly attributed to gonococcus infection. However, numerous other microorganisms of relatively mild virulence have also only a limited viability in the tubes. Only the tubercle bacilli and a few highly virulent strains of streptococci are able to survive for unlimited periods in the pelvic structures.

The histologic pictures of salpingitis are also not specific for any one type of infection. The claims of Schridde that gonococcus infection leaves characteristic pathologic changes within the tubes have been disproved by later investigators. It is commonly accepted that streptococcus infections ascend through the lymphatics to the parametrium and eventually to the tubes. However, Frankel observed cases in which streptococcus infection had apparently invaded the tube along the mucous membrane from the uterus, producing pathologic changes identical with those commonly ascribed to the gonococcus. With the growing experience that certain non-specific infections can produce pathologic and clinical pictures similar to those due to the gonococcus, it has been necessary to qualify the earlier notion that practically all cases of salpingitis result from gonococcus infection.

The English surgeon, MacKenzie, found that in his hospital near Manchester, less than 40 per cent of salpingitis cases were produced by gonococci, and the pure or mixed cultures of pyogenic cocci were more commonly responsible. Polak claimed that not more than 40 or 50 per cent of tubal infections are produced by gonococcus. In a large number of women on whom he operated for sterility, he found mild chronic tubal inflammations which could not be traced to any gonorrheal infections. In quite a few of these cases other pyogenic cocci could be found in the prostatic secretions of the partner. He thought it likely that non-specific pyogenic bacteria in the prostate of the husband can set up an infective endocervicitis in the wife, harmless in the majority of cases except for a constant, mucopurulent discharge. In such a case, any manipulation involving the cervical canal is apt to produce an endometritis and salpingitis.

There is no reason to doubt that, like gonorrhea, this type of infection can also spread spontaneously during menstruation, due to the accompanying lowered immunologic resistance, the physiologic congestion, and the favorable cultural medium of menstrual blood.

The literature shows numerous case reports of non-gonorrheal and non-puerperal salpingitis occurring even before the menstrual age. Riedel collected five cases of salpingitis of unknown origin causing a

fatal peritonitis in children under 10 years. Mazel reported the removal of a pus tube in a 10 year old child in whom repeated smears and cultures for tubercle bacilli and gonococci were negative. Caussade and others described salpingitis cases due to colon bacillus. There are a few case reports of parametritis and salpingitis following measles and scarlet fever, and quite a few had apparently a causal connection with influenza and the common cold.

Recent observations of Edward Allen and others show that acute pelvic inflammation may be associated with adnexal swellings, temperature and leucocytosis in a number of cases exhibiting trichomonas vaginalis. These attacks usually follow the menstrual period, but repeated smears and complement fixation tests for gonococci were negative. However, Allen and his co-workers isolated from the vaginal secretions associated with trichomonas a strain of streptococci which they believe responsible. The same type of organisms could be isolated from infected Bartholin glands in other trichomonas cases. They are inclined to think that pelvic cellulitis and salpingitis secondary to trichomonas vaginalis infection is a more common occurrence than is generally supposed. And they believe it not unlikely that the associated streptococci may ascend the genital tract of women almost as readily as gonococcus.

DIFFERENTIAL DIAGNOSIS

The frequently experienced difficulty in making a differential diagnosis between acute salpingitis and appendicitis is even more pronounced in cases of non-specific salpingitis in which the bacteriologic and serologic tests for gonorrhea are negative, and when no history and no signs of exposure are present.

Of the numerous points which differentiate acute salpingitis from acute appendicitis, I want to emphasize a few of the most significant.

An important characteristic in the history of salpingitis is that the majority of cases start subsequent to or during the latter part of a menstrual period. The period in question is most often described by the patient as having shown some unusual features as to the time of its arrival, length of duration, amount, or concomitant distress.

In acute appendicitis, a beginning with upper abdominal pain is the rule, in salpingitis the exception.

Of clinical signs, vaginal and rectal examination in acute salpingitis show tenderness of the fornices, usually on both sides and behind the cervix; a certain resistance of the parametrium may be felt, but no adnexal tumor in the initial stages. Attempts to move the cervix are painful.

Temperature, leucocyte and differential counts differ more with the type, virulence, and stage of the infection than with the localization in tube or appendix.

The most reliable guide in the differential diagnosis has recently been proven to be the blood sedimentation time. According to experiences of Lesser and Goldberger in a large series of cases, the sedimentation reaction offers a distinct aid in the differential diagnosis between acute salpingitis and acute appendicitis, salpingitis giving a consistently abnormal blood sedimentation reaction, appendicitis a consistently normal one. Harold Brunn confirms, through experiences of his own, the importance of the blood sedimentation test for the most difficult differential diagnosis between pelvic appendicitis and salpingitis. According to C. T. Smith and others, the increased rate of sedimentation is most significant during the first twenty-four to forty-eight hours after onset of symptoms in acute salpingitis, as compared with relatively normal sedimentation time in acute appendicitis.

But even when all the differential points which distinguish acute salpingitis from acute appendicitis are known and considered, there still remain a few atypical cases in which a differential diagnosis is impossible to make, and a diagnosis of appendicitis either is made or cannot be excluded with safety. Exploration in such

cases is not only justified, but occasionally unavoidable. In a large series of cases with the final diagnosis of salpingitis, Polak and Farr and Findlay found that 10 to 15 per cent had been operated upon with a preoperative diagnosis of appendicitis.

CASE REPORT

I want to report briefly a case of non-specific salpingitis in a girl of 17 years. Menstrual history was negative. She complained of gradually increasing dull pain in the lower right quadrant for approximately two weeks. It had started several days after an apparently completely normal menstruation. The pain was always localized. There was no nausea, vomiting or constipation, and no urinary symptoms. There was no distention; temperature and pulse were normal; urine negative; leucocyte count varied between 8,000 and 13,000, with a relative lymphocytosis. Sedimentation tests were not taken. As the pain became increasingly severe and the evening temperature started to rise, the patient was taken to the hospital for observation.

On the morning of her arrival, temperature was 98, pulse 80, white count 8,600 with 72 per cent polys. The patient felt quite comfortable. During the day pain increased, her temperature rose to 100.8, her pulse to 90, white count to 13,000 with 70 per cent polys; catheterized urine was negative. Clinical examination showed localized tenderness centering in an area about 5 cm. right of the umbilicus. There was slight rigidity and rebound tenderness. Since the patient had a virginal introitus, no vaginal examination was made. A moderate amount of whitish, thin vaginal discharge was observed but not examined at this time.

Rectal examination showed a normal sized uterus in anteversion; the adnexa could not be made out and no masses were palpable. Movement of the uterus was mildly painful, and there was a questionable tenderness in the right fornix. A diagnosis of an acute inflammation of a posteriorly located or retrocecal appendix was made, and operation decided upon.

On opening the abdomen some serous exudate was found. The appendix was normal and no Meckel's diverticulum was present. The right tube was swollen, tortuous and hyperemic, and its mucosa red and glistening. The right ovary and parametrium were congested. The left adnexa could not be brought into the field of vision and presented no changes appreciable through palpation. The appendix was removed, the abdomen closed without drainage, and the patient made an uneventful recovery.

During the hospital stay and later on, repeated smears from the urethra and from the region of the cervix were made; these showed no gonococci, but numerous Gram-positive and some Gram-negative cocci and bacilli. In one fresh smear trichomonas vaginalis was found. The final diagnosis was "non-specific catarrhal salpingitis."

As far as I know, the patient has been well since leaving hospital.

COMMENT

I believe that the case described was one of the number in which the correct diagnosis of acute salpingitis cannot be made with certainty and a laparotomy is unavoidable. On account of the chronic course and the mildness of the infection, blood sedimentation tests would have been of little help in this particular instance.

In the meantime I had occasion to use the sedimentation test in a number of similar but more acute cases, and found it eminently reliable and of great practical value for differentiating between acute tubal infection and appendicitis.

In such cases as the one above described, even after the abdomen is opened, the inflammatory changes produced by the initial stages of acute salpingitis are not pronounced enough to be appreciable by palpation alone; the diagnosis has to be ascertained by vision, and eventually by culture of the tubal secretion. This may be one of the reasons why acute catarrhal salpingitis is seldom diagnosed in the numerous cases where a small abdominal incision is made for the removal of a suspected appendix, although the appendix is found to be not responsible for the symptoms. Possibly it would be more often diagnosed if in such cases the incision would be extended far enough to permit a look at the adnexa.

SUMMARY

Non-specific infections are responsible for acute and chronic salpingitis in a considerable percentage of the cases.

They can present a history and produce clinical and pathologic pictures similar to those of gonococcus infections. The resultant pathologic changes in the tubes often cannot be distinguished from specific lesions.

Non-specific salpingitis can occur without previous exposure and may be added to the swelling list of pelvic disturbances found in young girls, often mistaken for acute or chronic appendicitis.

There are unquestionably a few cases in which the differential diagnosis between acute salpingitis and appendicitis cannot be made with certainty, and where laparotomy becomes imperative.

If exploratory laparotomy reveals an acute tubal inflammation instead of the expected appendicitis, the best procedure theoretically would be to close the abdomen without further interference. Usually, however, the appendix can be removed without harming the patient. This procedure has also the practical advantage of saving surgeon and patient a possible repetition of the same dilemma of differential diagnosis and possible second operation.

REFERENCES

Allen, E. Trichomonas vaginalis vaginitis. *Am. J. Surg.*, 33: 523–528 (Sept.) 1936. Clinical and bacteriologic observations in trichomonas vaginitis. *Am. J. Obst. & Gynec.*, 30: 565 (Oct.) 1935.

Bourne, A. Prognosis of salpingitis. *Lancet*, 2: 331–332 (Aug.) 1935.

Brunn, Harold. Acute pelvic appendicitis. *Surg., Gynec. & Obst.*, 63: 583–590, 1936.

Caussade, G., and Vignuel. Salpingitis due to the colon bacillus in a child 12 years old. *Bull. Soc. d'obst. et gynéc.*, 23: 659 (Nov.) 1934.

Cruikshank, G. C. in Teacher's Manual of Obstetrical and Gynecological Pathology. London, 1935. Oxford University Press.

Curtis. Bacteriology and pathology of Fallopian tubes removed at operation. *Surg., Gynec. & Obst.*, 33: 621–631, 1921. Chronic pelvic infections. *Surg., Gynec. & Obst.*, 42: 6–9, 1926.

Farr, C. E., and Findlay, R. T. Salpingitis. *Surg., Gynec. & Obst.*, 49: 647 (Nov.) 1929.

Frank, R. T. Gynecological and Obstetrical Pathology. New York, 1922. D. Appleton & Co.

Jones, W. J., and Bunting, C. H. Invasion of the Fallopian tube by oxyuris vermicularis; report of a case. *Arch. Path.*, 11: 229, 1931.

Lesser, A., and Goldberger, H. A. The blood sedimentation test and its value in the differential diagnosis of acute appendicitis. *Surg., Gynec. & Obst.*, 60: 157–166, 1935.

Levine, M. H. Management of the infected tube. *Med. J. & Rec.*, 137: 242 (March 15) 1933.

MacKenzie, J. A. Salpingitis and its treatment. *Practitioner*, 134: 217 (Feb.) 1935.

Mazel, M. S. Hematosalpinx and pyosalpinx in a child. *Illinois M. J.*, 62: 84–86, 1932.

Miller, C. J. The rational treatment of tubal disease. *Surg., Gynec. & Obst.*, 45: 110 (July) 1927.

Peightal, T. C. The treatment of salpingitis. *Journal-Lancet*, 55: 69 (Feb.) 1935.

Polak, J. O. Pelvic Inflammation in Women. New York, 1923. D. Appleton & Co.

Royster, H. A. The pus tube and its management. *Am. J. Obst. & Gynec.*, 10: 392, 1925.

Rumpf, W. H., Jr. Treatment of salpingitis. *Minnesota Med.*, 14: 1028–1030 (Dec.) 1931.

Smith, C. T., et al. Sedimentation time as an aid in differentiating acute appendicitis and acute salpingitis. *Am. J. M. Sc.*, 189: 383–387 (March) 1935.

Worral, R. Infections of the Fallopian tubes. *M. J. Australia*, p. 146–47, 1931.

Wu, L. C. Chronic salpingitis caused by oxyuris vermicularis; report of a case. *Chinese M. J.*, 49: 256–59, 1935.

Young, J., and Macgregor, T. N. The palliative treatment of salpingitis. *Edin. M. J.*, p. 129 (Oct.) 1932.

A MODIFIED STURMDORF TRACHELOPLASTY

LOUIS DROSIN, M.D.

Attending Gynecologist and Obstetrician, Beth David Hospital

NEW YORK CITY

DURING the past nine years I have been doing tracheloplasties by a modified Sturmdorf technique. It differs from the standard method essentially in the fact that instead of transverse suture aspects being opposed and united with transverse suture aspects, in my technique, longitudinal suture aspects are apposed and united with longitudinal suture aspects. The practical differences and advantages are that in the longitudinal method, better results are obtained, because: (a) the inverting flaps, in view of their tapering points, can be drawn up to a somewhat higher level in the cervical canal than can be done with the transverse ends in the classical Sturmdorf method, thus coapting the surfaces more thoroughly; (b) the lower stitch further precludes the possibility of any loose space where blood might accumulate or whence hemorrhage might take place; and (c) its execution is simpler and easier than the Sturmdorf method.

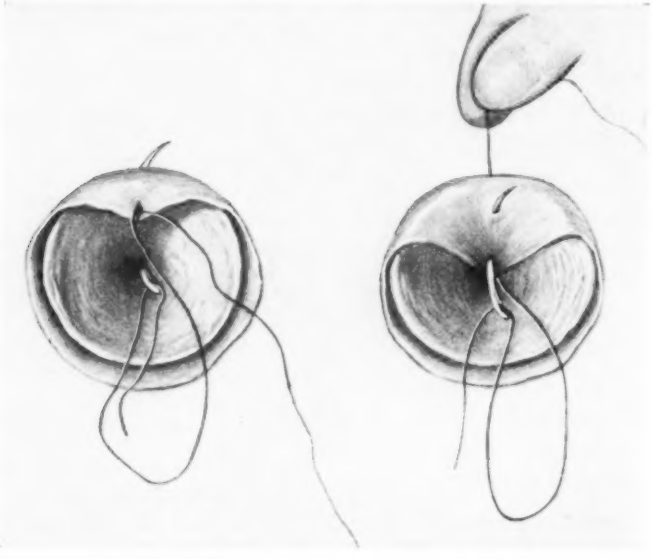


FIG. 1. FIG. 2.

FIG. 1. Suture inserted at central point of flap, one-quarter inch or less from the edge and tied at its under surface. The end proximal to the cervical canal is now in the course of being passed through the canal at the level of the internal os to come out on the vaginal surface. (While this is being done, the flap is supported and traction made on it with an Allis forceps; this is not shown in the illustration.)

FIG. 2. First suture passed and held taut while the lower one is being passed about midway between the internal os and the newly formed external os.

Technique. A cone is removed and a flap prepared as in the Sturmdorf method. Even after a curettage has been done, one must make sure that a sufficiently raw surface is created in the region of the internal os. This is accomplished with a scalpel used in a scraping rotary motion a few times. A suture is then passed through a central

point in the flap (anterior or posterior) one-fourth inch or less from the edge, and the ends tied at its undersurface (Fig. 1),

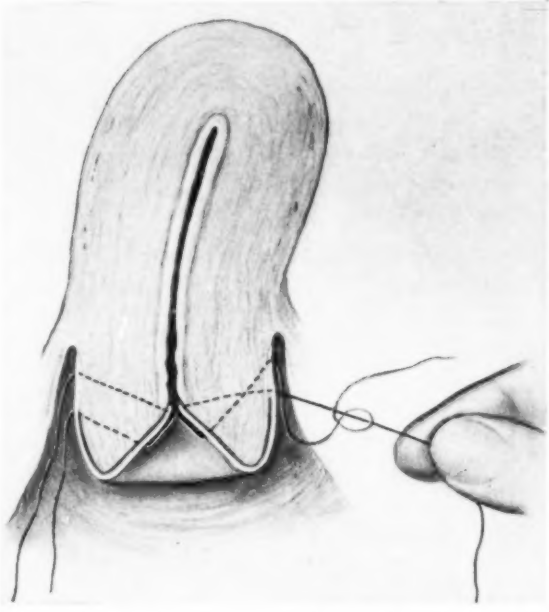


Fig. 3. Posterior segment shows the upper and lower sutures in place ready to be tied. Traction is to be made on the upper end with one hand while the other ties the knot. If traction is made on the lower or on both ends while tying, the desired result will not be obtained. Anterior segment indicates how traction on the upper or first suture, while inserting the second one, will cause the latter suture to come out higher than the first one. In this case traction must be made separately on both ends to determine which one invaginates the flap more thoroughly. The one that accomplishes this is held taut with one hand, while the other hand ties the knot.

both ends being left sufficiently long and uniform. While the flap is held steady with an Allis clamp (not shown in the illustration), the threaded end proximal to the cervical canal is inserted into the canal high up at the internal os and coming out on the vaginal surface. (Figs. 1 and 2.) While this end is being held taut (Fig. 2) so that the tip of the flap completely invaginates, the other threaded end is inserted similarly, but halfway between the internal os and the newly formed external os (Fig. 2), likewise coming out on the vaginal surface. When both flaps are thus treated, traction is made on the upper suture with one hand while the other hand ties the knot. (Figs. 2 and 3.) Most of the time, particularly when a cervical wedge is removed with the apex terminating at the internal os, that is,

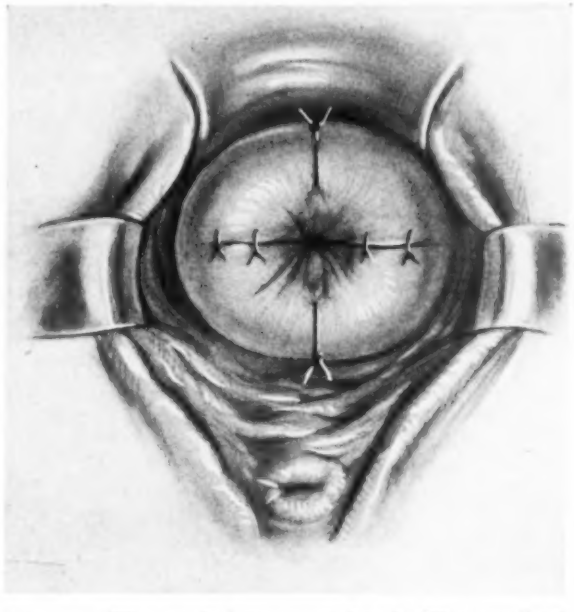


Fig. 4. The technique completed. The cardinal sutures are running in a longitudinal direction.

without leaving or making a shelf, and without making a flap as in the Sturmdorf technique, the second suture, while traction is being made on the first end, will come out higher on the vaginal surface than the first. (Fig. 3, anterior flap.) (This constitutes an important technical correction which cannot be done in the Sturmdorf method.) In this case, before attempting to tie the ends, it is necessary to use traction separately on both ends to determine which one inverts the flap more thoroughly. The one that does this better should be held taut with one hand while the other hand ties the knot. (Fig. 3.) It is needless to add that the flaps must be loose enough to facilitate invagination. The lateral sutures, when they are necessary, are inserted in the conventional manner. (Fig. 4.) Figure 4 also shows the cardinal suture appearing in a longitudinal instead of a transversal direction.

With this technique I obtain results as close as possible to a normal cervix. No complications, immediate or remote, have

been observed in operations performed in this manner.

SUMMARY AND CONCLUSION

A modified Sturmdorf tracheloplasty wherein longitudinal suture aspects are united with longitudinal suture aspects instead of transverse suture aspects. It has the advantage over the Sturmdorf method in that the inverting flaps can be drawn to a higher level at the internal os, thus precluding the possibility of loose or bleeding spaces which might cause hemorrhage or disunion. Such preclusion is enhanced by the reinforcing middle suture.

The removal of the cone-shaped wedge without leaving sufficient tissue for a regular shelf is particularly indicated in deep and diffuse infections, permitting more of the diseased tissue to be removed; the employment here of the longitudinal suture technique yields better results than the Sturmdorf operation.

The execution of the modified technique is simpler than the Sturmdorf method.

THE operation for the plastic repair of a hard or soft palate cleft should not be performed before eighteen months and better results and a lower mortality are obtained by waiting until the second or third year, or even later.

From—"Pediatric Surgery" by Edward C. Brenner (Lea & Febiger).

RECONSTRUCTION OF DEFORMED CHIN IN ITS RELATIONSHIP TO RHINOPLASTY

DERMAL GRAFT—PROCEDURE OF CHOICE

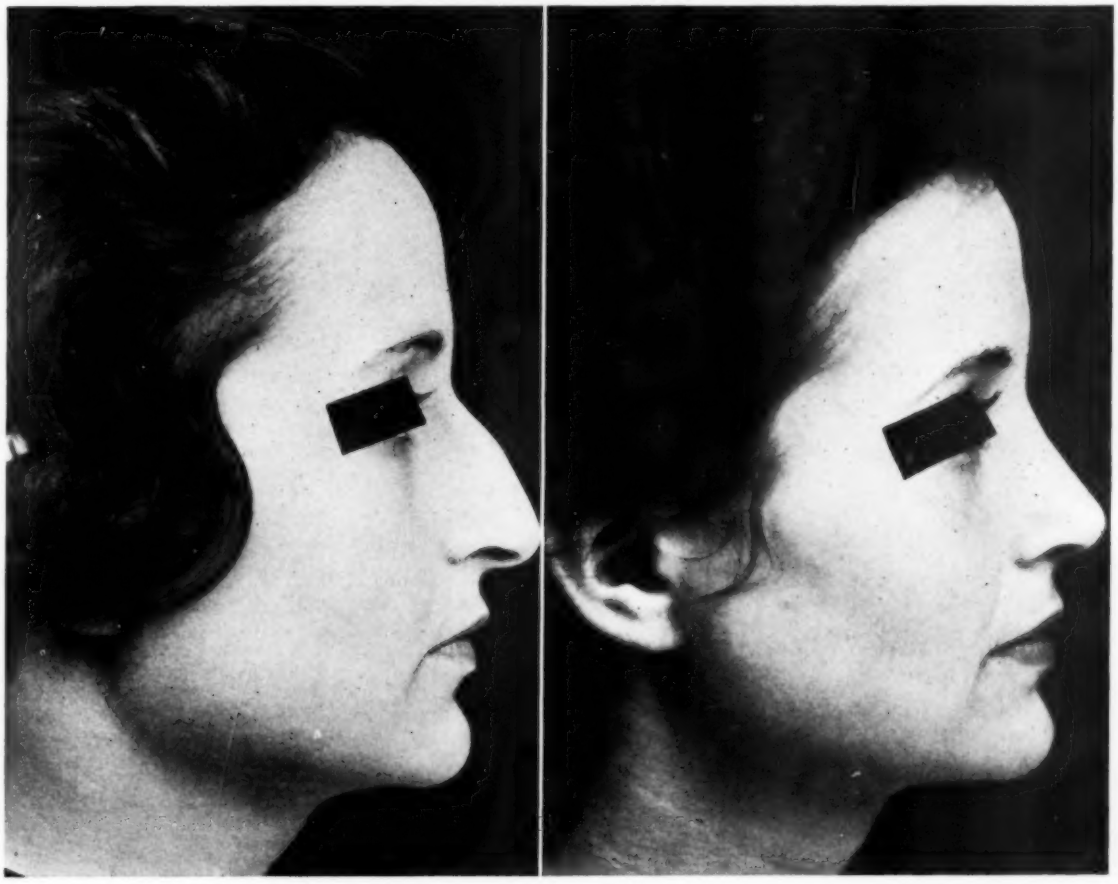
JACQUES W. MALINIAC, M.D.

Attending Plastic Surgeon, Sydenham and Jewish Memorial Hospitals

NEW YORK CITY

CORRECTIVE rhinoplasty has come to be one of the most common procedures in reparative surgery. With its growing popularity, however, attention is being focused on concomitant abnormalities of other components of the face and their relationship to the nose.

A reconstructed nose should not merely be inconspicuous; it should harmonize with the rest of the profile, particularly the forehead and chin. Failure to observe this principle frequently results in a faulty post-surgical profile which is a source of much dissatisfaction to the patient.



A B

FIG. 1. A profile definitely marred by an oversized nose in combination with a normally developed chin. Corrective rhinoplasty alone established normal proportion in profile. A, before and B, after operation.

In outlining the correction, a sketch is often helpful. On it the surgeon can study the possible changes, visualize the future profile and determine the effect improvement of the nose will have upon the contour of the face.

Although it is obviously impractical to attempt to lengthen or shorten the fore-

head, rebuilding of the chin coincidentally with reduction of the nose is frequently indicated.

discussion of chin deformities without attendant functional disturbance.

The chin occupies the front plane of the

A B

Fig. 2. Oversized nose and receding chin in girl of seventeen. Dermal graft used in rebuilding the chin. Rhinoplasty would not have been sufficient to establish an esthetic profile. A, before and B, after correction.

The most common types of chin deformities are the receding and the small or underdeveloped chin. In the majority of cases, these malformations are of congenital origin, but, a depressed chin may also be the result of injury, bony trauma in early childhood or scar formation. Scars around the chin, especially those which extend from chin to chest, cause pressure and interfere with normal bony development.

The chin abnormality may be traceable to pathologic factors by involvement of the jaw bones and teeth or ankylosis of the temporomandibular joint and malocclusion. Such malformations however, are comparatively rare and will not be covered here. Our report is confined chiefly to a jaw and may be angular, rounded, protruding or receding; it may be overdeveloped or underdeveloped. Normally the chin falls underneath the vermilion border of the lower lip. The upper surface of the chin, which marks its depth, is convex and follows into the center of the lower lip. Thus the shape, form and size of the chin lend the face a characteristic expression of strength or weakness.

PROCEDURES IN USE

Normally the chin consists of a firm fatty cushion of varying size and shape, imposed upon the jaw bone. The material to be used for reconstruction must fulfill the local requirements, that is, it should be pliable

and of proper consistency. Where there is definite deficiency of bony development, a cartilaginous or bony graft is indicated. In the receding but otherwise normal bony chin, a soft graft is more suitable to complement the normal consistency of the padding. The choice of the plastic material is of importance in view of the cosmetic effect.

Some surgeons employ the ivory implant.[1] A dish-shaped ivory prothesis with multiple perforations is introduced through a small incision in the lower surface of the chin and maintained in a tunnel in front of the bony jaw. The implant is maintained in place partly by means of fibrous tissue growing into the perforations.

In my opinion, the ivory implant is unsuited to this purpose. For one thing, its hardness does not conform to the natural consistency of the chin. Principally, however, here as elsewhere an autoplastic graft should always be given preference when possible.

While an iliac bone can easily be shaped to rebuild an underdeveloped chin, it is contraindicated in cases with a scanty elastic padding. The ease with which a rib cartilage graft may be shaped to fit the curvature of the chin renders it more suitable for this purpose than a bone graft. An additional advantage is its greater resistance to infection and absorption.[2] I have used rib cartilage in a number of cases with gratifying results. In many instances, however, the solid consistency is a decided drawback. The following is a case in point.

Several months after insertion of a rib cartilage graft, the patient, a young girl, complained of its hardness to the touch. Although she was satisfied with the post-surgical shape of the chin, she complained that her fiancé, unaware of surgical correction, commented on the hardness of the chin. This young woman became obsessed with the idea that the surgical camouflage might one day be discovered, and she insisted that the cartilage be removed and replaced by a softer material. We did so, employing a dermal graft. This was the last case in which we used cartilage for a chin reconstruction.

Where rhinoplasty is done at the same time as the chin correction, some surgeons advocate use of the osteocartilaginous hump removed from the dorsum to rebuild the chin. This method calls for the presence of a hump of considerable size, which is not always available. The procedure therefore seems feasible only in limited cases.

Fig. 3. The receding preoperative chin indicated in dotted lines. Dermal graft shown in sold line, placed anteriorly to chin to produce natural protuberance. There was no indication for lengthening the chin.

The fat graft has long been in use for filling out depressed areas on the face, it does not, however, possess the ideal requirements for an autoplastic material. Its great fragility and marked tendency to absorption make it difficult to determine the necessary degree of overcorrection.[3]

Dermal grafts, comprising a full-thickness of skin without epidermis have been used abroad for many years in general surgery for the closure of hernias, reinforcement of joints, etc.[4] In the past few years

A B

Fig. 4. Young girl of nineteen with a scoliotic, oversized nose and a markedly underdeveloped receding chin, resulting from difficult labor. A horizontal tunnel was formed in front of the jawbone, through a small incision made in the natural fold on the lower surface. The size and shape of the cartilaginous graft were prepared according to pattern. The cartilage, with perichondrium attached to one side, was made to fit the curvature of the jaw. The tunnel containing the cartilage was closed with chromic catgut to prevent displacement. The oversized nasal deformity was corrected in the usual way. A, before and B, after correction.

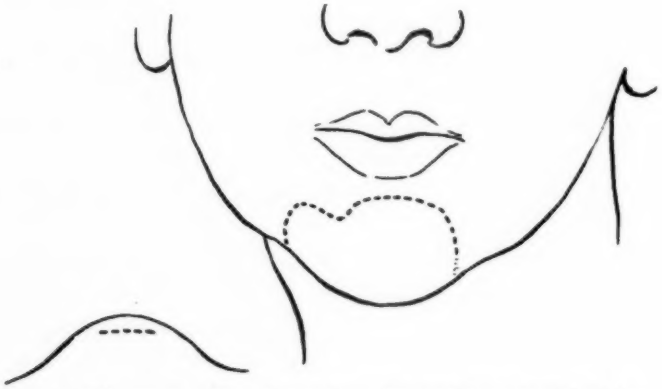


Fig. 5. Diagram at left shows location and length of incision through which a dermal or cartilaginous graft is inserted. The irregular shape of rib cartilage graft in upper sketch corresponds to asymmetry of chin as shown in photograph (Fig. 4).

I have used dermal grafts for filling out facial depressions with invariably good results. In my opinion it is the substance of choice in upholstering the facial contour. When inserted subcutaneously it preserves its integrity with minimum shrinkage and no untoward complications.

Our main objection to dermal grafts at first was the possibility of cyst formation due to the subcutaneous inclusion of skin appendages (hair follicles and glands). Experience proved, however, that these appendages atrophy and disappear after a time.[5]

Favorable experience with this type of graft in other facial defects led us to use it for building up the receding and undersized chin. Our results so far have been highly satisfactory to both the patients and ourselves.

A strong argument in favor of the dermal graft is its accessibility and greater resistance to infection. In a few cases we have seen dermal grafts partially exposed to infected wounds, heal in place without apparent damage.

The dermal graft can easily be prepared in the required size and shape and, if necessary, can be superimposed in layers. The graft shrinks slightly in time (10 to 15 per cent) and for this reason moderate overcorrection is indicated. However, there is much to recommend its use in preference to the fat graft, where a 50 to 60 per cent loss by absorption is not uncommon.

The nature of the material permits of exact affixation, thus eliminating the possibility of displacement of the graft in the reconstructed part. There is no tendency to lobulation, as in the fat graft; the transplanted derm becomes evenly incorporated in the surrounding tissues, showing a firm structure.

CONCLUSION

Reconstruction of the undersized chin coincidentally with reduction of the nose is often indicated, to establish a harmonious profile.

The most common types of chin deformities are the receding and small, or underdeveloped chin.

A cartilaginous graft is indicated in reconstruction of the underdeveloped chin; dermal graft is the substance of choice for the receding type.

REFERENCES

1. Joseph, J. Nasenplastik und sonstige Gesichtplastik nebst Mamaplastik. Verlag Curt Kabitsch. Leipzig, 1931, p. 695.
2. Maliniac, Jacques W. Cartilage and ivory—indications and contraindications for their use as a nasal support. *Arch. Otolaryngol.*, 17: 649–657 (May) 1933.
3. Maliniac, Jacques W. Comparative value of surgical procedures in repair of skin defects. *Med. J. & Rec.*, Jan. 21, 1931.
4. Rehn, E. Das cutane and subcutane Bindegewebe in veranderter Funktion. *Arch. f. klin. Chir.*, 105: 1, 1914.
5. Lowe, O. Jahres berichte des erztichen Vereins zu Frankfurt A/M. 1913–1914.

THE CAUSES OF SURGICAL FAILURE IN CLEFT PALATE OPERATIONS

IRVING WILSON VOORHEES, M.S., M.D.

Assistant Surgeon, Manhattan Eye, Ear and Throat Hospital

NEW YORK CITY

A SURGEON is one who *looks in* and *finds out*. When he has ceased finding out, he has ceased to be a surgeon. "Experience," said Hippocrates, "is fallacious and judgment difficult." But, we may add that *experience*, based on a well cultivated habit of reflection, is the only sure way in which accurate *judgment* can be brought to bear the fruit called *wisdom*. We need, too, not only our own experience for the building up of knowledge, but that of other men in whom we have confidence, for our own experience may be so limited that it is practically worthless, but when added to the testimony of others, it may become crystallized into something useful. It was with these facts in mind that the present study was undertaken.

Blair of St. Louis, in his article on cleft palate in the National Encyclopedia (Collier & Co.) says that "staphylorrhaphy is one of the most difficult operations in all surgery." By this I feel sure that he does not mean the mere process of cutting and suturing, but the matter of getting a satisfactory result when healing is completed. Technically, the operation is not difficult—certainly not so difficult as submucous resection of the nasal septum in an old fracture, for example—but for many years, poor results have made the operation tabu to most general surgeons, and to not a few who restrict their surgical activities to work about the head. Some operators seem to have had a feeling that attempted repair of the palate is something worse than a waste of time. "Why bother with it?" said one, "You know you can't do these children any good."

It is with a view to finding out what a cross section of opinion might show if it could be obtained from those surgeons who do such work frequently, that I felt it worthwhile to "sound them out" as to the causes of this alleged failure. Accordingly, personal letters were sent to a selected list of men practicing this sort of "plastic" in various large medical centers throughout the United States and Canada. Inasmuch as the questionnaire method of obtaining such information has been done to death, it was thought better to ask surgeons freely to express their opinions and experience, instead of setting questions for them to answer. The response was generous in the extreme; about 98 per cent of replies were received, which shows the great interest this topic arouses. Several of the correspondents wrote *in extenso*, and asked that, if they had not made their position quite clear, they be queried further to elucidate moot points. I would be more than ungrateful if I did not here express deep thanks for this splendid coöperation. The response was inspiring, and will, I believe, be definitely helpful. The following letter from Dr. G. V. I. Brown of Milwaukee illustrates the coöperative spirit which obtained:

"I am very much interested in the fact that you have undertaken to collect data for the study of the causes of failure following cleft palate operations, because I feel that you have taken up a phase of the subject which ought to have been studied in a systematic way long years ago.

"As a matter of fact complete failure following staphylorrhaphy ought to occur very seldom.

"In my private work, and in the clinics of which I have charge here at the Milwaukee Children's Hospital and at the Wisconsin General Hospital at Madison, we rarely ever

have a failure insofar as securing closure of the palate fissure may be concerned, and then, usually, because of something we have left undone that we ought to have done.

"I no longer look in the mouths of my patients constantly as I formerly thought I had to do, because we feel very sure of the results, and the less youngsters are disturbed the better.

"Judging by my own past misfortunes, and the results of previous operations by others as the patients have come to me later for correction, I would enumerate the causes of failure as follows:

"1. Failure to overcome and guard sufficiently against tension.

"2. Insufficient blood supply for the flaps.

"When the Von Langenbeck-Warren mucoperiosteal operation was performed, as I used to do it for many years, there was in some cases a more or less sufficient blood supply from the anterior vessels through the incisive foramina and the posterior palatine vessels, but raising the flaps by making an incision along the inner borders of the fissure took away the blood supply, and in cases where the tissue was not thick and well nourished, a partial failure through the formation of an opening in the center of the palate was unavoidable; unless great care was taken, failure of the sutures to hold the parts together was very likely to be much more certain.

"Looking back over the years, I feel that that operation made a good showing.

"The operation as I now perform it, by the formation of bone flaps, closing the posterior portion of the palate first, and one year later the anterior portion by raising mucoperiosteal flaps which are, however, small because the remaining anterior portion of the opening is usually very small, has given me results that I have never been able to obtain in any other way.

"The reason is that the plentiful blood supply from the nose is undiminished and the same blood supply that the other operations have depended upon is also effective here.

"Not only is mortality almost nil, but we do not have the high temperatures and the very sick babies we used to have.

"With the child in good condition and a plentiful blood supply, the mucous membrane of the mouth will clear itself usually very satisfactorily with but little postoperative treatment of any kind.

"On the other hand, when a child's general condition is poor, if for example, there is a tendency to acidosis, bacteria and secretions cling to the mucous membrane. Then, even the greatest care cannot clear the operative field sufficiently to keep the parts in good condition.

"3. Traumatization of tissue.

"Operators unaccustomed to working in the mouth always cause more damage to the tissue than experienced oral surgeons.

"I have seen operators damage tissue in the formation of flaps, so that looking on, one could be almost sure that these could not survive.

"4. Tying sutures too tight.

"Unless the parts can be brought together freely without tension, any attempt to hold them with tightly drawn sutures must fail. The sutures cut and pull out.

"5. The condition of the patient at time of operation.

"The child must be in as perfect condition as possible. I send every child from the table without operation who comes there with any condition out of the ordinary. If the trouble passes, they are brought back again. I know that I reduce mortality and also the number of sick postoperative infants that I would otherwise have.

"I congratulate you on the efforts you are making to investigate this feature of palate work, and would appreciate being informed as to the results of your research."

AGE

A wide diversity of opinion exists as to the age most favorable to securing a satisfactory result. This may be classified as follows:

(1) Do early; (2) too early; (3) too late; and (4) early and late.

The *early* operators advocate a range from seven days to two years. H. A. Potts would do the operation in the first week, or during the first two or three months. He agrees with the majority that harelip, if present, should be corrected before the palate operation, and that there should be a waiting period before the latter procedure, according to the will of the operator.

Risdon of Toronto likes to have the cases in hand somewhere between the

eighteenth and twenty-fourth month, while Baldwin of Columbus, Ohio, thinks that one year strikes a good average.

The *too early* objectors are notably Blair, Dorrance, Vaughan and Dunning. The latter says, "Most of the operations that are failures either are done too early or are badly planned." And Dorrance affirms that, "The older a child is, the thicker the tissues over the hard palate are," meaning by inference that thicker tissue is tougher tissue and more likely to withstand tension.

Ladd of Boston speaks of the undesirability of "postponing the operation until *too late* in life." This opinion is well supported by common experience and seems to be based upon the poor vascularity of the tissue commonly present in later life, upon the inadequate blood supply to the flaps, and upon a relatively insufficient amount of tissue which can be "borrowed" to close what is, in adults, a wide gap with narrow margins.

Early and late: Ivy of Philadelphia is the only author who happens to mention the range of age during which he has operated successfully. This covers a span from 2½ to 27 years! He uses the Veau and the Dorrance "push-back" technique.

"In this connection," says Dr. Herbert A. Potts of Chicago, "I think 2 or 3 months of age is the best time to operate a cleft. I do the lip first, or, if there is no cleft lip, I do the palate the first week, if I can.

"If I think the mucoperiosteum too thin, I pack it with vaseline gauze for two or three weeks, then operate on it. The patient should be in the best physical condition, with blood normal and gaining in weight.

"I am very careful not to traumatize the tissues during operation; to get no tension on the flaps, and to have irrigation after feeding, as well as before and after operation.

"I try to have the babies in the hospital three or four days before operation.

"To avoid a short palate I draw the palate upward and backward by means of nasally placed sutures passing through the pharyngeal muscles."

GENERAL CONDITION

As might be expected, practically all operators lay stress on good general health as a prerequisite to successful repair. This holds especially for the ward cases, most of whom come from poor families and have, therefore, never known or enjoyed the regimen of food and living which those in better circumstances usually have had. Federspiel builds up the general condition for some time prior to operation, through food tonics and general hygienic measures. Risdon speaks of a "sugar diet" supplemented by a "milk mixture." He is probably thinking of younger children. Potts states that "the blood must be normal." I assume that he means a normal hemoglobin, differential and red cell count with a negative Wassermann. He is the only correspondent who mentions this extremely important item, but the others no doubt require blood examinations as a routine measure which is a part of the hospital "work-up." Vaughan, Davis and Brown emphasize good general health.

INFLUENCE OF HEALTHY RESPIRATORY TRACT

All membrane adjacent to the mouth should be as free as possible from infectious organisms. This dictum might be well summed up in the postulate: *Avoid infection, both pre- and postoperative!* Lyons states that "pediatrics plays a big part in the success or failure of this operation." Vaughan studies the nose and throat as well as the mouth, and is on the lookout for Vincent's organisms in any of these areas. He speaks of the importance of knowing the history of the individual as to high temperature, low resistance and the infectious diseases of childhood, especially of measles. Risdon names coryza and infectious diseases as enemies to success, and Blair puts the surgeon to the foil when he speaks of the "breath of the operator" as a source of postoperative

infection. Dunning sums the matter up in two words, namely: "avoid infection."

Prophylaxis of the nose, mouth and throat would seem to be a *sine qua non*, but the methods of bringing this about are not described by any of the correspondents. Federspiel says "Clean up the teeth and gums." Potts commands: "Prepare the patient for three days in the hospital before operation." Dunning avers that inexperienced operators are prone to use faulty methods, and that poor or bad planning makes for almost certain failure. Blair follows the plan of the late Dr. John E. Mackenty who warned against operating upon patients during the winter months when infections are prevalent. Since there is no emergency feature present, spring and summer may be recommended, and the reasons therefor stated when talking over the plans with parents who sometimes make up their minds suddenly and are likely to urge immediate operation while they are "in the mood."

Says Dr. William Ladd of Boston:

"Taking failure of union first, I would consider respiratory infections or middle ear infections as the most important and probably the commonest causes of failure of union. I state this because I find that patients who are in a private room have very few failures. I think I could take the last one hundred *private* cases and show that not over one or two have partially pulled apart. Neither the results of my associates nor my own results in the charity ward are as good as this. Though it is extremely uncommon, even in the wards, to have a complete failure of union, the last time I checked up there were something over 10 per cent of the cases who developed a small hole at the junction of the hard and soft palate, or a slight failure of union anteriorly. Not many of these hampered the eventual outcome, but a second operation was required. After respiratory infections, I consider the most important causes of failure of union to be interference with the blood supply by destruction of the posterior palatine vessels, or by tying the sutures too tight, or using some form of tension apparatus. The latter we gave up many years ago. The flaps should be so free that tension apparatus is not required.

"Poor functional results I believe are largely due to failure in extending the lateral incisions far enough back, or to postponing operation until too late in life. I have done a few palate operations in the second decade which looked to me like splendid anatomic results, but the patients have never been able to speak worth two cents. The other bad functional results I see are in patients who have had two or three unsuccessful operations elsewhere, causing excessive scar tissue and preventing a flexible or mobile soft palate even after union is attained."

SOCIAL CONSIDERATIONS

Theoretically, there should be a difference in results according to the social status, a point which has already been touched upon, but several surgeons seem to think that this makes little difference. For example, Ladd obtains "good results" in 98 per cent of his private patients and 90 per cent in the ward cases. Vaughan, of New York, states: "I have almost completely eliminated failures by avoiding, as much as possible, the above factors." He names age, physical condition, presence or absence of infections, especially of Vincent's and measles, and faulty technique as the factors.

Dunning (New York) expects 95 per cent success in all of his patients, but it is assumed that he selects them with care. Dr. Dunning writes as follows:

"I am doing most of this work at the Medical Center and would say that we have very few failures in cleft palate operations. We ought to expect to get about 95 per cent success in the first operation. Most of the operations that are failures are either done too early or are badly planned; too much is attempted at one operation which is roughly executed; there is too much tension on the sutures; injury to the

blood supply; infection; poor postoperative care; and operations by the inexperienced who perform them only occasionally, and therefore are not quite up to the job."

TECHNIQUE

In the minds of most persons, laity and surgeons alike, there is a deep-seated feeling that "technique" is responsible for success in all operative work. That is, I think, a little too far reaching, for it exacts more from brain and hand than these should reasonably bear. Every surgeon feels sure that *his* technique is as perfect as it is humanly possible to make it, but, in the nature of things there is no 100 per cent of success over a period of years in any field of endeavor. We must deal with unknown elements, no matter how rigid our preparation, and surprise and disgust sometimes come to us from indeterminate sources.

However, there seems to be one text from which every surgeon wishes to preach a sermon, namely, *avoid surgical trauma!* These words occur again and again in the writings of all authorities on cleft palate surgery.

A second text might be propounded with the words, *conserve blood supply!* Occasionally, however, so little is thought of the importance of this dictum, that a recommendation is made to "cut the posterior palatine arteries." For example, Dorrance, who has written an excellent book on cleft palate surgery, does his operation in two stages. In the first stage he "divides the posterior palatine arteries"; in the second, he "waits for a thickening of the flaps." This seems to be another way of saying that after severing the arteries, one must wait for a reëstablishment of collateral circulation in the flaps before utilizing them. Only those who have performed his "push-back operation" may pass judgment on this, but many who follow him, notably Burdick of New York, are enthusiastic. This was true of that excellent rhinolaryngologist, the late Dr. George Carroll of Rochester, who recommended and performed the Dorrance operation in all cases.

As to conservation of blood supply, Dr. Chalmers J. Lyons (University School of Dentistry, Ann Arbor, Michigan) says: "We are emphasizing the conservation of all of the blood supply of the posterior palatine vessels. In the past, we have thought that there was an anastomosis between the posterior palatine vessels and the anterior. We have come to the conclusion after considerable investigation that, if there is an anastomosis, it is in the anterior palatine canal and does not come down onto the palate. We believe that the entire palate is supplied with blood from the posterior palatine. We have made some nice dissections to show this and to show that right at the junction of the hard and soft palates there are very few collateral vessels, and we believe that the reason one gets these post-operative openings is that there is a lack of blood supply to that part."

Federspiel (Milwaukee) is a conservative who opposes long lateral incisions, and he is upheld in this by many others. Ivy, of Philadelphia, uses the method of Dorrance of the same city, and also, at times, the Veau (Paris) technique.

Other advocates of conservation of the blood supply are notably Ladd, who cautions against an "interference with blood supply by destruction of the posterior palatine vessels," Federspiel who has seen "starvation necrosis from injured blood supply," Risdon (Toronto) who inveighs against the "cutting of arteries and destruction of the blood supply," and Dunning, who avoids "injury to the blood supply," without being specific.

As to "denudation" and preparation of the soft parts preliminary to actual suturing, Ferris Smith, of Grand Rapids, "splits the margins of the cleft and does little or no denudation." He relies on the placement of sutures to bring the raw edges together and thus effect permanent union. Davis, of Philadelphia, on the contrary, thinks that "failure is often due to insuffi-

cient denudation" and Blair of St. Louis blames "insufficient freeing of the tissues" for some failures.

However, the seemingly radical difference in these factors is not so great after all, for everyone knows that in order to get any union between mucous membranes, one must have a "raw" surface. The variation seems to depend upon the manner of making the surface raw. It is, of course, absolutely necessary to conserve every millimeter of tissue in a region where the width of the gap to be filled in often seems larger than the amount of "fill" to be obtained. Federspiel thinks that, "Failure following cleft palate operations is largely due to errors in technique, especially so when the circulation is disturbed sufficiently in the palatal tissue to cause starvation necrosis."

Then, of course, we must also take into consideration the resistance of the patient, and any systemic involvement that is a factor in preventing healing. Infections also play an important part, especially with patients who have decay of teeth, gingival pericementitis, periapical abscesses, etc.

SUTURES

Slight mention is made of the kind of suture material used by the correspondents. It seems to be taken for granted that either silk, horsehair, or linen is inevitable. Older writers were careful to explain that these fillets must be left in place much longer than the traditional five days allotted to skin incisions. Why? The limited experience I have had, supplemented by watching the postoperative progress in patients operated upon by others, leads me to believe that the purpose of sutures is served by the fifth day. Thereafter, a suture is a foreign body and, as such, is likely to *delay* healing rather than to *promote* it. Removable or non-absorbable sutures are not unattended with the risk of breaking down a part of the healing line, whether removal be done with or without general anesthesia. The stitches are likely to be encrusted with secretions from the wound edges plus inspissated nasal and oral mucus, and are not always visible, since, even when black thread is used, it is decolorized within a few days.

What about chromic gut for this purpose? Personally, I have found it highly satisfactory, and feel sure that it is used by many operators who never emphasize the fact. The twenty-day variety of small size, number one or smaller, never larger, should be chosen. It should be softened by the nurse in order that its "looping" tendencies do not become an annoyance to the operator who already has enough to worry about in his hour and one-half of concentrated effort.

By the fifth day there is either cross union, or a pulling away of the edges of the midline. The latter denotes that the wound margins are healing together, but not across the gap as desired. The fillets are fairly loose by this time and are doing very little to keep the tissues in approximation. If gut has been used, it can be removed without difficulty, as it is partially digested and may not even require to be cut with scissors.

Regarding removal of sutures, Lyons, of Michigan, does this on the fifth day; F. Smith of Grand Rapids, on the sixth or seventh; and Logan, of Chicago, on either the ninth or eleventh day according to conditions. All operators are unanimous in urging that no tension be permitted, that sutures must not be tied too tight, and that inaccurate approximation must be avoided. Some do not favor lead plates or other means of taking the strain from the midline. It is difficult to find any good ground for objecting to this, for upon avoidance of tension on the suture line depends primary union or the lack of it. In retrospect, it seems to me that the Mackenty technique in this particular is not only serviceable but indispensable, and after following his cases, I decided to use lead bands in every instance.

Just a word about this. Small rectangles of lead about 1 inch wide, 3 inches long and ½ inch thick are procured, usually through

a hardware dealer. When needed they are hammered to the thinness of an ordinary sheet of paper. There is no good reason why they could not be rolled out in a machine, but we still seem to cling to this primitive method. They are sterilized by boiling for a half hour and may then be cut with scissors as required. Stab incisions are made through the soft palate on each side, just avoiding the posterior palatine vessels as determined by the hamular process which may readily be felt by the finger. These stab incisions are stretched with a hemostat until it is possible to pass a lead strip about 1 cm. wide through the incisions from side to side. The ends are not yet cut off, but the strip is kept from slipping out of place by sutures passed through and held to the cheek by ZO tape.

Another lead strip, ½ inch wide, is inserted through the lateral incisions about the center of the hard palate area. We are now ready to place the sutures. After they are tied, the ends of the lead strips are sewn together, leaving about ¼ inch of the ends hanging free. This is of great value in keeping the tongue away from the suture line. Dr. Mackenty used a sort of basket-work applied to the teeth to keep the tongue away. This is quite unnecessary, as the sharp or rough edges of the lead strips soon educate the tongue to avoid the discomfort occasioned by this contact.

Dr. Fulton Risdon (Toronto) says:

"I, perhaps, use an older method, and that is lead plates over wires in the posterior palate region on each side, which I remove on the tenth day, not for tension, but for splinting the palate. My belief is that the long lateral incisions predispose to loss of circulation and failure. Further, it is very difficult to splint the palate with such long incisions. I am well aware that the lead plates have been given up quite largely by most men, but I still continue to use them with great satisfaction."

POSTOPERATIVE CARE

In general, it seems better not to do too much postoperative meddling. But the mouth as a whole should be kept as clean as possible, consistent with no disturbance of the suture line. Ferris Smith (Grand Rapids) believes in a policy of "hands off," while Dunning (New York) thinks that poor postoperative care is the cause of some failures. Davis (Philadelphia) believes in "cleansing the suture line" as do several other correspondents.

Whatever is done should be carried out by the operator himself. It should not be delegated to a nurse or house officer, for this is no place to divide responsibility. Some prefer saline irrigations to nose and throat, avoiding contact with the sutures. It is doubtful whether this is possible. Certainly no "scrubbing" with a bit of cotton or gauze should ever be done, for it does no good at all, and is a bid for failure. Personally, I drop a solution of 50 per cent hexylresorcinol through the nose and along the suture line of the oral side, but the tip of the dropper never comes into contact with anything. Maybe it does no good, but I feel that it does no harm. In this connection, the method of feeding is of great importance. The end of a spoon or the nipple of a milk bottle, even nursing at the mother's breast, may spoil our work. It is better to give liquid food only through the nose via a soft rubber catheter which is passed close to the septum and rather high in the nose away from the palate, the tip reaching just to the oropharynx. Only a few drops should be sent through at a time so that coughing or strangling will not take place. This is a laborious procedure, but should be carried out for the first five days in all cases of children under 5 years of age. Thus given, food does not contact the suture line to form a nest for bacteria.

Dr. Vilray P. Blair of St. Louis says: "I think the following are causes for failure in cleft palate operations: too early operations, insufficient freeing of the tissues; inaccurate approximation with sutures; destruction of blood supply by cutting the lateral arteries; and, most common of all, infection.

"Infection can come either from an ordinary cold, or most commonly, from the breath of the operator. I think we have

somewhat lessened our infections *by not operating on cleft palates between the first of January and warm summer weather*, since that is the time upper respiratory infections are most prevalent. The interoccurrence of measles, whooping cough, pneumonia, etc., will interfere with healing. Any child who has a temperature over 103°F. two days after operation is apt to have a hole in the palate.

"Any man who reports no failure either does not operate on many palates or takes liberties with the exact truth."

GOOD OPERATIVE RESULT WITH BAD FUNCTION

Not infrequently, a fine surgical result is associated with a stiff palate, excessive scar tissue and rhinolalia; therefore, the importance of educating the palate muscles to their new duties in the formation of correct speech must be emphasized. This is another reason for doing the operation before speech habits are formed, for it is so difficult to change habits that are deep-seated or long continued. Baldwin (Columbus, Ohio) says that, "If done after talking age, the operation results in poor speech." This is a common observation; therefore, speech training is imperative in every case, whether the operation be done early or later. There are, in most towns of any size, teachers who work privately or in connection with public schools, who take great interest in this matter of speech improvement and who are very assiduous and successful in obtaining results. It is, to be sure, a highly specialized form of pedagogy, requires no end of patience, and is commonly not well rewarded. All honor to those teachers who toil so indefatigably in helping these children to a normal place in society.

In olden times, it was thought that cleft palate children were stigmatized not only through physical defection, which is always apparent to the ear after speech is attempted, but also through mental defection as well. Nothing can be further from the truth. Many of these unfortunaet children are bright, wholesome and affectionate. However, they are soon made to feel their "unlikeness" to normal children, and therefore develop an inferiority complex which makes them seem stupid, slovenly or ill-tempered, and consequently, is likely to make them socially dependent. A successful operative attack may convert them into fine, upstanding, independent citizens. Is there a more worthy objective in all surgery? Certainly, it is far more likely to be successful than many of the operations on the brains of mental defectives who must, of necessity, be substandard, no matter how self-righteous the well intentioned surgeon may feel in his effort to bring forth salvage from the human rubbish heap. Staphylorrhaphy is now on a sound surgical footing, and this little investigation brings to light the intense interest of the oral plastic surgeon, and the joy he feels in his great work.

SUMMARY

Many physicians feel that attempts to repair a palate cleft are a waste of time because of poor results. There are, of course, some failures in spite of improved technique. What are the causes of these failures? To determine these, personal letters were sent to the leading men who do this work.

A cross-section of opinion seems to point to: improper selection of cases; operating in the presence of recent mucous membrane infections; cutting off of blood supply to flaps; incomplete preparation of "raw edges" of flaps; improper suture material; improper placing of sutures; too much meddling after operation, or neglect of proper postoperative care; good anatomic result with poor function; lack of speech training when healing is completed.

In general, the results are much better than they were twenty-five years ago. Some operators have, in fact, almost a perfect score. It is to these men that we must look for guidance in perfecting our technique.

OPERATIVE MANAGEMENT OF TRAUMATIC INTRACRANIAL HEMORRHAGE*

E. S. GURDJIAN, M.D.

Attending Neurosurgeon, Grace Hospital; Associate Attending Surgeon, Receiving Hospital; Extramural Lecturer, Graduate Medical School, University of Michigan

DETROIT, MICHIGAN

ALTHOUGH the majority of patients with head injury do well on conservative treatment, a goodly number present signs and symptoms that may spell operative intervention. With the present day tendency of over-conservatism in the management of head injury it is important to evaluate such signs and symptoms and effect operative intervention as soon as it is justifiable or necessary. It is always a painful experience to note at autopsy a pathologic condition which could have been treated by operation with fair degree of probability for a successful outcome.

The pathologic possibilities in traumatic intracranial hemorrhage are given in the following tabulation:

I. Intracranial hemorrhage
   - A. Extradural, due to rupture of meningeal vessels, sinuses and diploe
   - B. Intradural, due to pial tears, bruises or laceration of nervous tissue
     - (1) Subdural
       - (a) Acute
       - (b) Chronic subdural hematoma
     - (2) Subarachnoid
       - (a) Generalized
       - (b) Localized
     - (3) Intraparenchymatous
       - (a) Petechial
       - (b) Massive

II. Bruising or laceration of nervous tissue, with or without fracture of the skull.

III. Subdural accumulation of spinal fluid simulating intracranial hemorrhage.

In the above classification of listed conditions best treated by operation are extradural hemorrhage, acute subdural hemorrhage with signs, chronic subdural hematoma and subdural collection of spinal fluid. Because of similarity of symptoms and signs, some cases on exploration may show only contusions and lacerations of neural tissue or a wet brain with no demonstrable gross pathology. It must be admitted that in a great many there may be a combination of lesions.

This paper is based on eighty-one operated cases during a five and one-half year period from September 1930, to March 1936. I have included in this group only those cases which were treated by myself, and have not attempted an all-inclusive statistical survey. The distribution of pathology is as follows: 19 cases with extradural hemorrhage; 11 cases of acute subdural hemorrhage; 13 cases of chronic subdural hematoma; 13 cases of subdural collection of spinal fluid; 15 cases of wet brain, cerebral contusions, subarachnoid hemorrhage (any one or a combination of all). Before discussing these pathologic entities, I should like to consider briefly the various signs and symptoms in head injury which spell operative intervention. These may be grouped under the following headings: first, changes in the state of consciousness; second, changes in vital functions; third, changes in pupillary size and ocular function; fourth, abnormal

* From the Department of Neurosurgery, Grace and Receiving Hospitals, Detroit, Michigan.

neurologic findings; fifth, changes in spinal fluid findings.

1. *Changes in the State of Consciousness.*

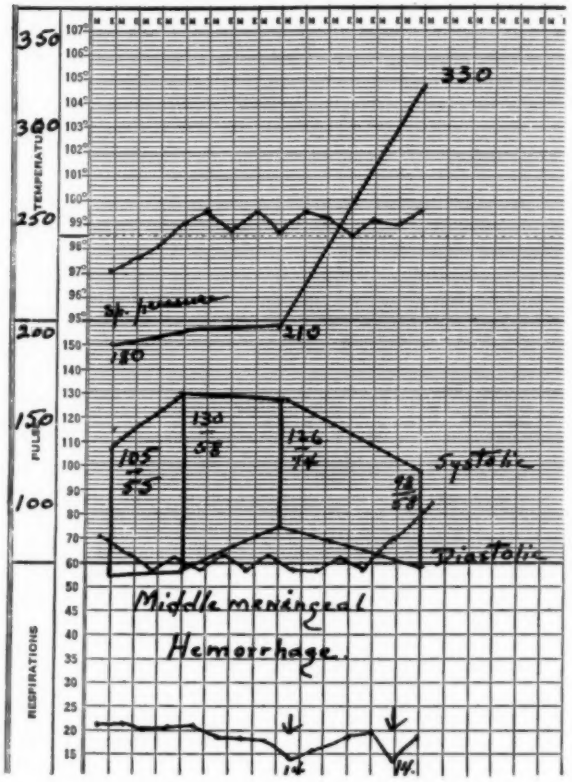


Chart 1. Composite chart of the vital functions and spinal fluid pressure in a case of middle meningeal hemorrhage. Note that blood pressure does not rise with increasing spinal fluid pressure. The respiratory and pulse rates became depressed on several occasions. The initial spinal fluid pressure in this case is low but this does not rule out middle meningeal hemorrhage of operable proportions.

Changes in the state of consciousness are very important. A normal state of consciousness is always a favorable sign and is a counterindication for operative intervention except in cases with mechanical defects (simple and compound depressions). Progressive return of consciousness is practically always a counterindication for operation. Progressive drowsiness, periods of conscious states alternating with periods of drowsiness, and disorientation may signify operative intervention, if certain other signs obtain. A lucid interval followed by drowsiness and unconsciousness is an important syndrome, but it does not always signify middle meningeal hemorrhage. I have seen it frequently with subdural hemorrhage, subdural accumulation of spinal fluid and edema of the brain.

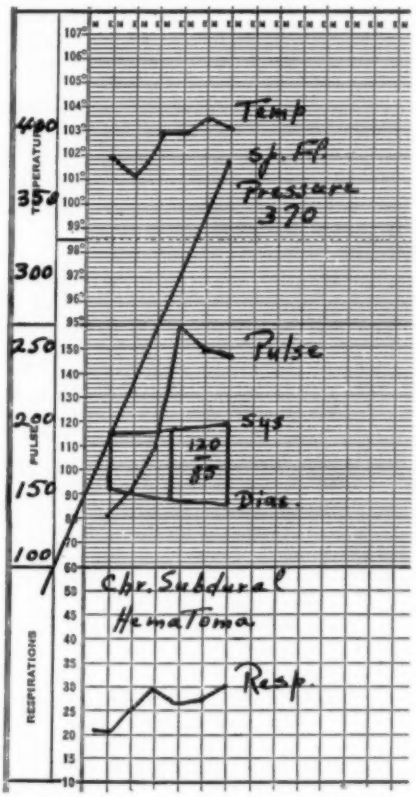


Chart 11. A case of middle meningeal hemorrhage with initial low spinal fluid pressure which became more depressed in the course of seventy-two hours, when the patient was operated upon. Even though a low spinal fluid pressure is unusual, it can occur in cases of middle meningeal hemorrhage. This is the only instance in the series showing a pressure under 300 at the time of operation. There is a blood pressure rise while the spinal fluid pressure is lowering. Blood pressure readings as an index of intracranial pressure seem worthless.

2. *Changes in Vital Functions.* The pulse, respiration, temperature and blood pressure are important both from a prognostic and a diagnostic standpoint. At times the pulse rate is low to an alarming degree. A low pulse rate is frequently associated with enlarging clots such as from extradural or subdural hemorrhage, although, a rate of 40 to 55 is occasionally seen with an otherwise normal patient

(including normal spinal fluid pressure). Such a case is kept under close observation for other signs of increased intracranial pressure. Usually the pulse rate rises to normal from a few to ten days. Of course the mere presence of a low pulse rate is not an indication for operation, but a lowering rate is an important factor in establishing its advisability. In conjunction with other findings (such as increasing drowsiness) it becomes a potent factor favoring intervention.

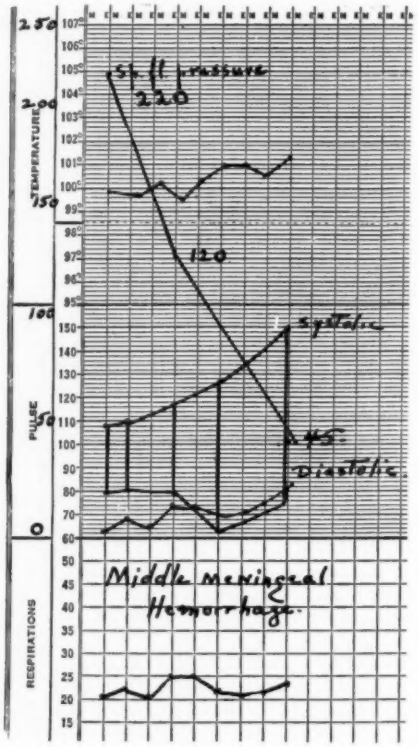


Chart III. A typical case of chronic subdural hematoma which has been allowed to go too long. There is increase in temperature, pulse and respirations. There is also a definite increase in the spinal fluid pressure. At this stage the patient is usually unconscious and may or may not have localizing signs. The presence of increased spinal fluid pressure in an otherwise normal person (except for the state of unconsciousness) is very suggestive of chronic subdural hematoma. A history of injury to the head may or may not be obtained.

The respiratory rate and the temperature are more frequently of prognostic significance. Increase in respiration soon after trauma spells very poor prognosis. High temperature (above 104 degrees rectally) soon after trauma, is also of serious import.

I have found blood pressure readings of no significant help in estimating the degree of intracranial pressure. On the whole, blood pressure readings are worthwhile only to establish the condition of circulation. One type of blood pressure reading has been found to spell very poor prognosis in the adult, *i.e.*, when the pulse pressure equals the systolic pressure.

3. *Changes in Pupillary Size and Ocular Function.* The condition of the pupils, their size, their reaction to light and inequalities, if present, are important both from a prognostic and a diagnostic standpoint. A dilated pupil is practically always on the same side as the lesion, for which an operation may be contemplated. However, in a small number of cases, the dilated pupil may be found on the opposite side, as was true in one of our cases of middle meningeal hemorrhage. Extraocular paralysis, when present, usually means pathology on the same side, when an operation is contemplated.

The prognostic significance of pupillary findings cannot be denied. Dilated and fixed pupils spell grave outcome. The same is to a less degree true in cases of constricted and fixed pupils. However, one can usually tell from the general appearance of the patient that he is in grave danger and such pupillary findings are therefore corroborative.

The presence of pupillary inequality as such is not a criterion for operation. When it appears in a case with other symptoms and signs favoring intervention, pupillary inequality helps to locate the pathology more accurately.

4. *Neurologic Findings.* Neurologic findings when present serve two purposes: first, the localization of pathology, and second, on the basis of the mode of development of such findings, they serve as indication for operative intervention. Hemiplegia, hemiparesis, Jacksonian convulsions, and

sensory changes, when detectable, usually denote pathologic changes in a definite portion of the nervous system. However, because of the great incidence of contrecoup lesions in head injury, neurologic findings of exact localization are at times quite misleading. Hemiparesis or hemiplegia on the same side as the cerebral clot is seen with sufficient frequency to warn the physician of its possibility. That such a state may be brought about is to be explained on the basis of either a contrecoup involvement of the cortex on the opposite side by pressure from the enlarging clot, or more frequently by contrecoup lesion of the cortex at the time of the accident.

In the greatest majority, the presence of a definite neurologic finding as such is not an indication for operation. The mode of appearance of said neurologic finding is much more significant. Hemiplegia seen immediately following an accident has a different significance from a hemiplegia developing slowly and becoming complete within twelve to twenty-four hours. In the former, the hemiplegia is most probably indicative of contusion or laceration of the motor cortex. When hemiplegia develops slowly, the possibility of an enlarging clot as a causative factor is apparent. Hemiplegia caused by contusion and laceration of motor centers appears immediately after injury. A combination of minor contusions of the motor centers and edema of the brain due to head injury may give the picture of a progressive paralysis. However, the spinal fluid in such a case is bloody and its pressure is not so high as in cases of extradural or subdural hemorrhage.

TABLE I

SYNOPSIS OF CASES WITH MIDDLE MENINGEAL HEMORRHAGE (OPERATED)

No.	Age	Pupils	Extra-Ocular Palsies	Lucid Interval	Pulse Rate	Focal Signs	Spinal Fluid	Results
1	55	Equal	None	None	50–60	Bilateral Babinski	Bloody 350	Recovered
2	13	At first unequal left larger	None	7 days with periods of disorient. No unconsc.	50–55	Right Babinski Right upper limb paresis	Bloody 180–300	Recovered
3	34	Right larger	None	None		Left hemiplegia		Recovered
4		Equal	None	None		None		Recovered
5	22	Constricted on same side	None	About 15 hr.	55–60	Right hemiparesis	Bloody 350	Recovered
6	34	Right larger	None	None	Normal	Left Babinski		Recovered
7		Right larger	None	None	55–60	Left hemiplegia	Bloody	Died
8	41	Left larger	Lt. 3rd	None	50–60	Right hemiparesis		Recovered
9	8	Right larger	Lt. 3rd	14–18 hrs.	55–60	Left hemiparesis		Recovered
10	26	Right larger	Rt. 3rd	12–14 hrs.	120–130	Bilateral signs		Recovered
11	11	Right larger	None	2 hrs.	50–60	Left hemiplegia Bilateral Babinski		Died
12	46	Right larger	None	Drowsy throughout	Normal	None		Recovered
13	54	Right larger	None	Drowsy throughout Disorient.	Normal	Left Babinski		Recovered
14	10	Left larger	Lt. 3rd	10–12 hrs.	45–60	Right hemiparesis	Bloody 600	Recovered
15	36	Right larger	None	31 days with periods of disorient.	Normal	Left hemiplegia		Recovered
16	53	Right larger	None	Drowsy throughout	60–65	Left hemiplegia		Recovered

Note that in three cases operation was performed seven, nine, and thirty-one days after accident, respectively. The pulse rate is uniformly low in the greatest majority. Focal signs were present in fourteen cases. Dilated pupil on the side of the lesion with paralysis of the opposite half of the body was present in thirteen cases.

5. Changes in Spinal Fluid Findings. Spinal fluid findings are very important. The blood content of the spinal fluid is of prognostic and diagnostic significance. Bloody spinal fluid does not rule out an extradural hemorrhage of benign propor-

tions. In all cases of middle meningeal hemorrhage in this series spinal puncture when performed yielded bloody fluid. Of course, when the fluid is markedly bloody, the possibility of associated brain damage with massive intracerebral hemorrhage should be kept in mind. Bloody spinal fluid does not rule out chronic subdural hematoma. In a goodly number the fluid was either bloody or xanthochromatic.

TABLE II

MIDDLE MENINGEAL HEMORRHAGE (OPERATED CASES)

State of Consciousness	
Lucid interval	6
Unconscious throughout	7
Drowsy with disorientation	3
Pupils	
Large on same side	13
Large on opposite side	1
Equal	2
Extra-Ocular Palsies	
Third	4
Fourth	1
Sixth	1
Focal Signs	
Present	14
Not present	2
Vital Functions	
Pulse	45–60
Respirations	18–26
Temperature	100–102
Spinal Fluid Findings	
Pressure	300–650
Bloody	In all 5 cases punctured
Results	
Recovered	14
Died	2

Note that in cases of middle meningeal hemorrhage, lucid interval occurred in not more than 40 per cent. There was one case with a dilated pupil on the opposite side. Third nerve paralysis on the same side as the clot occurred in four patients. Focal signs were present in fourteen. Respirations and temperature were within normal limits, usually with a low pulse rate, certainly in the beginning. Spinal fluid pressure was uniformly high and the spinal fluid was bloody in each instance. The presence of bloody spinal fluid should not deter operative intervention in cases of middle meningeal hemorrhage. The tendency to attribute the clinical findings in the case to subarachnoid hemorrhage is to be deplored.

Spinal fluid pressure obtained by water or mercury manometer is frequently of deciding significance. The pressure is always high particularly early in operative conditions excepting where operation is indicated for mechanical defects (simple and compound depressions). Fluid pressure of 300 and over is significant. In cases where the spinal fluid is definitely bloody with pressure varying between 200 and 300, the possibility of cerebral contusions and pial tears causing the increased fluid pressure is apparent. Such pressure invariably drops down as the patient slowly recovers. In case of an operable condition, the pressure stays up or goes higher, except when the patient is very ill. A pressure of 150 is compatible with middle meningeal hemorrhage. This was true in one of our cases.

Extremely high spinal fluid pressure (750 plus) tends to become lower in patients who are progressively getting worse, as is well brought out by Browder and Meyers. At times a subnormal spinal fluid pressure is obtained in a patient who is evidently fatally ill. It is difficult to explain such low pressure, particularly in the presence of marked cerebral damage as shown by bloody spinal fluid. Possibly it is due to a deranged circulatory mechanism in the nervous system even though the general body circulation may be working at par or above par.

In patients who remain drowsy or disoriented for days or weeks, the presence of high spinal fluid pressure (300 and over) is a strong argument in favor of operation. In such cases, chronic subdural hematoma and occasionally an extradural clot may be uncovered.

Summary of Signs and Symptoms. It is evident from the above discussion that a combination of signs and symptoms may spell operative intervention. For example, low pulse rate of itself is not as significant as when associated with increasing drowsiness. With constant attention to the patient's condition and its varying manifestations, one usually arrives at a sane conclusion as to the proper procedure. It should be emphasized that each case of head injury should be individually treated on its own merits. There is sufficient variation in the manifestations of the pathologic processes to make a routine policy worthless. This will become more evident in the following discussion of extradural hemorrhage, acute and chronic sub-

dural hemorrhage, subdural collection of spinal fluid and certain cases of contusions and lacerations of the brain.

EXTRADURAL HEMORRHAGE

Extradural hemorrhage may be brought about by bleeding from bone, sinuses, meningeal vessels and emissary veins. Diploic bleeding is not usually of sufficient size to cause symptoms. We have seen fairly large extradural clots from rupture of the occipital emissary vein with depressed fracture in this vicinity. In none were there evidences of pressure symptoms from the clot. As a rule a discussion of extradural hemorrhage implies the group with bleeding from the middle meningeal artery. In the present series sixteen out of nineteen cases had rupture of the middle meningeal artery, two had rupture of the anterior meningeal and one was associated with tear of the lateral sinus.

In the typical case, the diagnosis of middle meningeal hemorrhage is easy. The usual story of head injury followed by a period of lucid interval which is then followed by progressive drowsiness, dilated pupil on the same side and evidences of paralysis on the opposite side makes the diagnosis a simple one. However, such a history may obtain with other than hemorrhage from the middle meningeal artery. We have seen this syndrome with acute subdural hemorrhage, edema of the brain and subdural collection of spinal fluid.

The absence of lucid interval is no argument against the diagnosis of middle meningeal hemorrhage and in some cases of benign outcome. The lucid interval is present if the hemorrhage is slow and there is no associated severe brain injury. It may be absent if the hemorrhage is very rapid, causing death two to three hours following the accident, or if there is associated severe brain damage. Marked disorientation alternating with lucid interval was observed in three cases. These people were operated on six, eight, and thirty-two days after the accident, respectively.

Dilated pupil on the side of the lesion associated with paresis or paralysis of the opposite half of the body was observed in twelve of sixteen cases. In three the pupils were equal and in one case the pupil was larger on the opposite side. In the latter, the hemiparesis pointed to the lesion on the side with the constricted pupil. Extraocular palsies were present in four of sixteen cases. It is reasonable to assume that the presence of such a palsy implies a more basal hemorrhage with the arterial tear in the vicinity of the foramen spinosum. With hemorrhage in this situation there is greater likelihood of implication of extraocular nerves as they course through the superior orbital fissure. Hemorrhage arising from rupture of the posterior branch of the middle meningeal artery compresses the lateral surface of the hemisphere and it is not usually associated with extraocular palsy. With tear of the posterior branch, there is greater likelihood of pupils equal in size. Hemiplegia or hemiparesis was present in fourteen of sixteen cases.

In an earlier communication, we discussed the similarity of Weber's syndrome (alternating oculomotor paralysis) and some cases of middle meningeal hemorrhage. This syndrome was seen in four cases. Each had third nerve palsy on the same side as the lesion with paralysis or paresis of the opposite half of the body. It is important to note this similarity, for the location of the lesion as described by Weber in cases of alternating oculomotor paralysis is in the midbrain, whereas, these cases of extradural hemorrhage proved beyond doubt that such a syndrome may be brought about by an enlarging clot above the tentorium. The third nerve paralysis is brought about by pressure against the third nerve in its course through the superior orbital fissure and the enlarging clot at the same time causes a paresis or paralysis of the opposite half of the body by pressing against the motor cortex.

Subdural hemorrhage caused by rupture of the middle meningeal artery internal to the dura was seen in one case. There was

also an extradural clot in this patient. The possibility of massive subdural hemorrhage from rupture of the middle meningeal artery internal to the dura is well admitted, such example but we have seen three others in cases which were not operated on. In one case, this temporosphenoidal clot weighed between 35 and 40 Gm.

TABLE III

OPERATED CASES OF CHRONIC SUBDURAL HEMATOMA

Case	Etiology	Ocular Changes	Fracture of Skull	Focal Signs	Location of Hematoma	Spinal Fluid	Operation	Results
1. C. G.	Auto	None	No	Aphasia. Left hemiparesis	Left		Left subtemporal decompression	Recovered
2. J. R.	Boxing	None	Yes	Right Jacksonian once	Left	350 clear	Bilateral trephine	Recovered
3. H. C.	Hit on head	None	Yes	Headaches, more to the left	Left	450 clear	Bilateral trephine	Recovered
4. J. D.		Right pupil dilated	No		Right		Right subtemporal	Recovered
5. F. F.	Football	Choked discs	No	None	Left	400 clear	Left trephine	Recovered
6. J. L.	Rocked on chair falling backward	Right pupil larger	No	None	Right		Right subtemporal Left trephine	Recovered
7. L. G.	Fight	Left 3rd	Yes	Right hemiparesis	Left	370 slightly bloody	Left subtemporal decompression	Recovered
8. E. N.	Auto	None	Yes	Left hemiplegia	Right	450 clear	Bilateral subtemporal depression	Recovered
9. J. O.	Auto	None	Yes	Left hemiparesis	Right	400 clear	Bilateral decompression	Recovered
10. C. R.	Shot	None	No	Aphasia, right hemiplegia	Left	Bloody increased pressure	Left subtemporal decompression	Recovered
11. E. D.	Fight	Left pupil larger	No	Suggestive right hemiparesis	Left	400 clear	Left subtemporal Right trephine	Recovered
12. M. O.	Fight	None	Yes	Right hemiparesis	Left	450	Left decompression Right trephine	Recovered
13. C. O.	Auto	None	Yes	Aphasia	Left	300 slightly bloody	Left subtemporal	Recovered

Among the operated cases of chronic subdural hematoma ocular manifestations occurred among five out of thirteen. There was fracture of the skull in seven cases. The site of the fracture does not necessarily indicate the side on which the clot may be found. The focal signs were frequent in this group but in a certain number some of these signs had disappeared completely by the time the patient entered the institution for treatment. For instance, although Case 2 had a Jacksonian epilepsy, this occurred only once and for several weeks he complained of severe headaches and at times became stuporous. The spinal fluid findings showed increased pressure in every one of the cases punctured. Xanthochromatic or bloody spinal fluid was obtained in three cases.

but this is undoubtedly a rare occurrence. A serious complication of middle meningeal hemorrhage is an occasional massive clot in the temporosphenoidal lobe on the same side. In the operated group there was one

The general appearance of the patient is of great importance. In the absence of severe associated brain damage, patients with middle meningeal hemorrhage usually do not look very ill. The pulse rate is almost

always low in the beginning. The respirations are not elevated and temperature reads between 100 and 102 rectally. The onset of symptoms is gradual, the patient showing more signs with every elapsed hour or more. The gradual onset of signs and symptoms is an important diagnostic criterion, although we may occasionally get the same picture with acute subdural hemorrhage or edema of the brain.

At times the position of fracture is of localizing significance, although in the greatest majority the diagnosis should be made on the basis of signs and symptoms. If the head is explored on the side of the fracture shown in the *x*-ray and no pathology is discovered, it is justifiable to explore the opposite side. The spinal fluid examination shows high pressure ranging between 300 to 650 by the water manometer. The fluid is bloody in each case punctured, usually mildly so. In one of our cases it was very bloody, but this patient had an associated intracerebral clot in the temporosphenoidal lobe on the same side. Slightly bloody spinal fluid or severely bloody spinal fluid does not rule out middle meningeal hemorrhage. All but one of the punctured cases recovered.

Table IV gives a summary of the cases as well as the signs and symptoms shown. Of great interest is the relative infrequency of lucid interval. It is also significant to observe that in a few patients disorientation alternating with periods of lucidity was the paramount change in state of consciousness. It may be assumed that among these, the bleeding is sufficiently slow to allow the brain to compensate for space occupied by the clot; hence the absence of motor phenomena and period of lucidity followed by unconsciousness.

TABLE IV

FOCAL SIGNS IN OPERATED CASES OF ACUTE SUBDURAL HEMORRHAGE, SUBDURAL COLLECTION OF FLUID, EDEMA AND CONTUSIONS OF BRAIN

	Acute Subdural Hemorrhage	Subdural Collection of Spinal Fluid	Edema of Brain Cerebral Contusions and Subarachnoid Hemorrhage
No. of cases	11	13	15
No. of deaths	5	6	8
Dilated pupils	5	5	5
Hemiparesis or hemiplegia	5	4	7
Convulsions			
Jacksonian	2	2	2
Generalized	0	0	1
Lucid interval	2	2	2
Continued unconsciousness and disorientation	4	4	
Dilated pupil with contralateral paralysis or paresis	4	2	3

It is to be noted that lucid interval occurred in two cases with each of the conditions above enumerated. Dilated pupil on the side of the lesion with hemiparesis or hemiplegia of the opposite half of the body occurred in four cases with acute subdural hemorrhage, two cases with subdural collection of spinal fluid and in three cases with edema of the brain, cerebral contusions and subarachnoid hemorrhage. Continued unconsciousness and disorientation was frequently seen among cases with acute subdural hemorrhage and subdural collection of spinal fluid. Jacksonian convulsions were frequent in this group. Although the signs seen in this group may simulate cases of extradural hemorrhage, patients with the latter condition usually look much better certainly in the earlier stages of the disease.

The operative technique and the methods used in the treatment of middle meningeal hemorrhage are removal of the clot through subtemporal decompression or osteoplastic flap. Either is preceded by an initial exploratory opening just in front and above or just behind and above the ear, or both. The use of a flexible brain

spatula of proper dimensions is worthwhile in that it may be introduced in the epidural space and thus a fairly large area may be

Fig. 1. Clot due to middle meningeal artery rupture obtained through an osteoplastic flap. With this procedure a mere complete evacuation of the clot is possible.

inspected. If necessary bilateral trepanation may be resorted to. This was necessary in one of our cases.

We have used general anesthesia more often, assuming that increased intracranial pressure caused by the anesthetic may accelerate the obliteration of the epidural space and minimize postoperative bleeding. Careful work is of course important, but speed is a definite criterion in the results obtained. These patients are usually on the verge of shock, and long drawn out operative technique will not help in the majority. After removal of the clot the bleeding vessel, if found, is ligated. We usually pack the epidural space loosely with iodoform gauze which controls bleeding and cuts down operative time. The packing is removed during the following two days.

CHRONIC SUBDURAL HEMATOMA

Although a history of minor head injury is frequent among cases of chronic subdural hematoma, the association of this condition with severe brain injury and fracture of the skull has been repeatedly described in the past few years. In the present group of thirteen cases, seven had fracture of the skull. Undoubtedly the reason for such preponderance of associated fracture in this group is due to the rather large fracture service at the Receiving Hospital in Detroit. When chronic subdural hematoma complicates skull fracture with brain injury, a long drawn out state of drowsiness, disorientation, or both may ensue. In such instances there may be drowsiness or disorientation for weeks or months, and patients may even die if the chronic subdural hematoma is not suspected or treated.

Focal signs were present in a great many in this group. Among ocular manifestations, dilated pupil on the side of the lesion was seen in three, third nerve palsy in one, and choked disks in another. Localizing signs were absent in four cases. The most characteristic finding was headache, and this was practically always lateralized. In two cases definite remissions occurred. The latter occurrence is significant in that chronic subdural hematoma is not always characterized by progression of symptoms and signs. In two cases in this series, the psychic manifestations were the most important symptoms. That patients with chronic subdural hematoma may be sent to insane asylums as mental cases has already been emphasized in the literature.

The spinal fluid findings were indicative of high pressure, ranging between 350 and 450 mm. of water. In three cases the spinal fluid was bloody. Bloody or xanthochromatic spinal fluid does not rule out chronic subdural hematoma.

As concerns cases of fracture of the skull who do not recover properly and remain

disoriented or drowsy for several weeks, the important thing to remember is that there may be a complicating chronic subdural hematoma. The most important diagnostic criterion in such a case is increased spinal fluid pressure. The site of fracture is of no localizing significance in that we have found the hemorrhage on the same side, on the opposite side, and in one case associated with an occipital fracture.

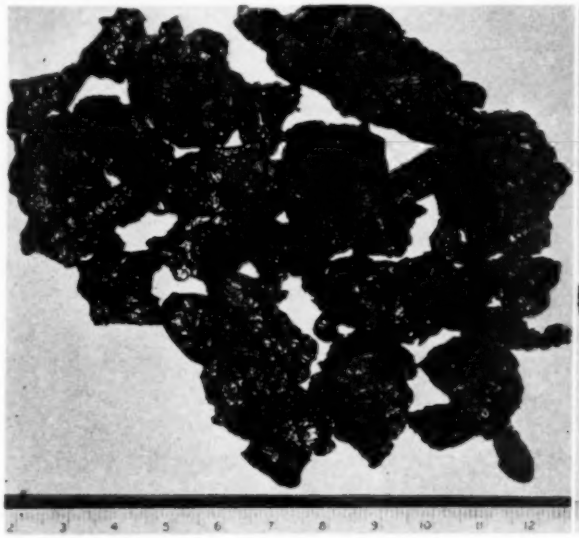


Fig. 2.

Fig. 3.

Figs. 2 and 3. Clots obtained from patients shown in Figures 6, 7, 8, and 9.

Fig. 4. Shows clot evacuated from epidural space thirty-one days after the accident. In this case the bleeding must have been very slow with gradual receding of the brain to allow for space occupied by the clot. Patient had left hemiplegia with periods of disorientation and drowsiness. The removal of the clot was followed by complete restoration of function to the left half of the body.

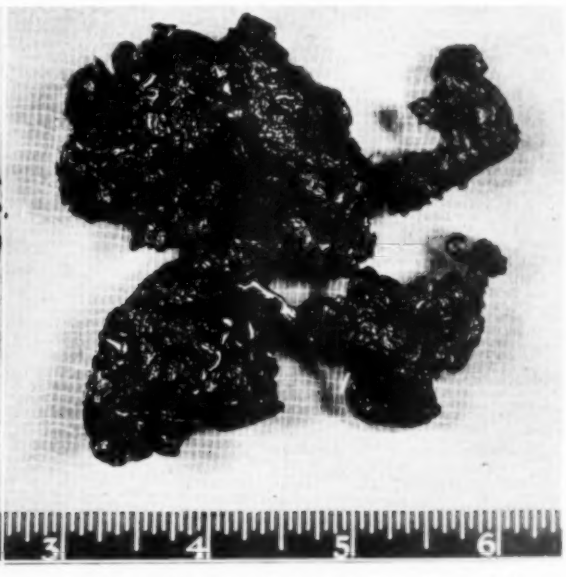


Fig. 5. This clot was obtained in a patient with a dilated pupil on the opposite side.

When chronic subdural hematoma is suspected, it may be ruled out by either encephalography or exploratory trephine openings on both sides of the skull.

Organization of the clot in the subdural space is begun immediately and evidences may be seen under the microscope in less than twenty-four hours. In a case of acute subdural hemorrhage from boxing, organi-

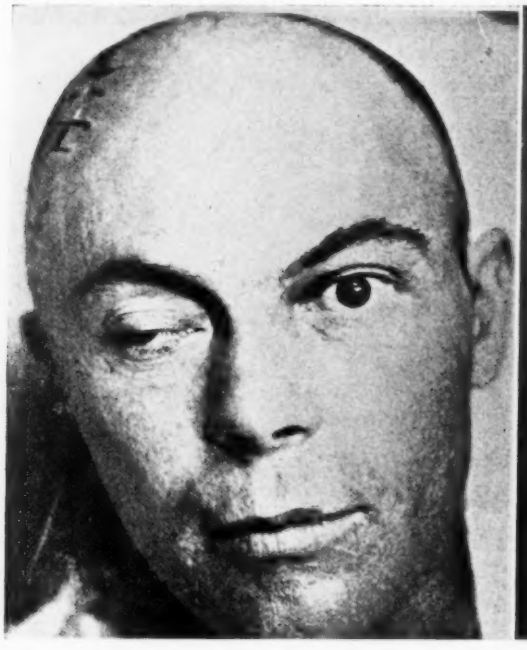


Fig. 6.

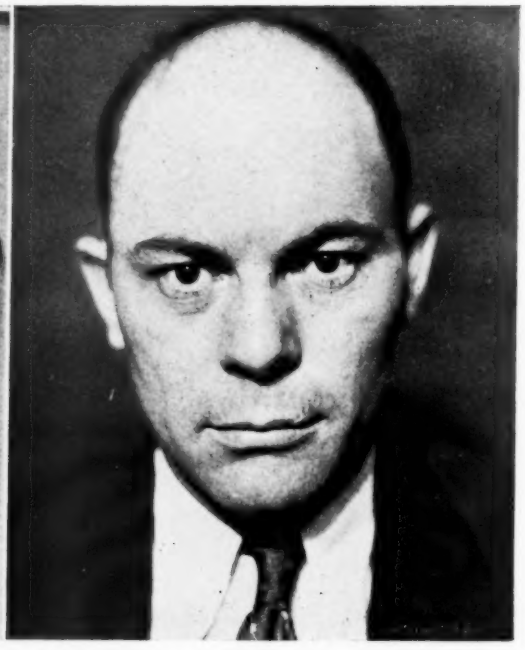


Fig. 7.

Figs. 6 and 7. A case of middle meningeal hemorrhage with third nerve paralysis. Recovery of oculomotor function usually obtains in three to five weeks after operation. The pupil on the affected side usually remains larger for a longer period. Eventually reaction to light and in accommodation obtains.

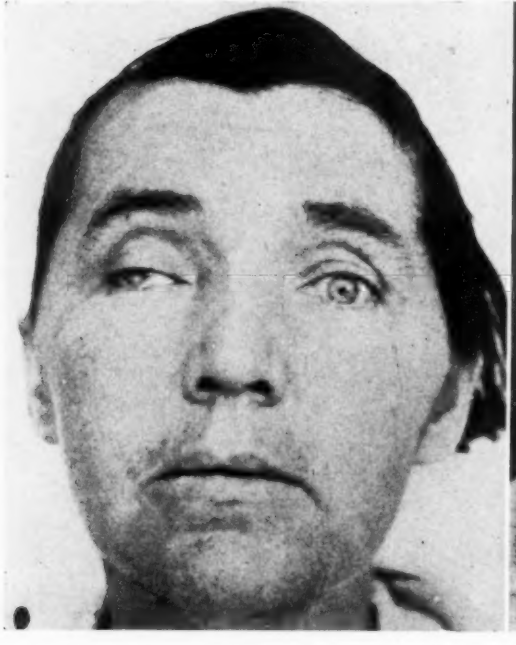


Fig. 8.

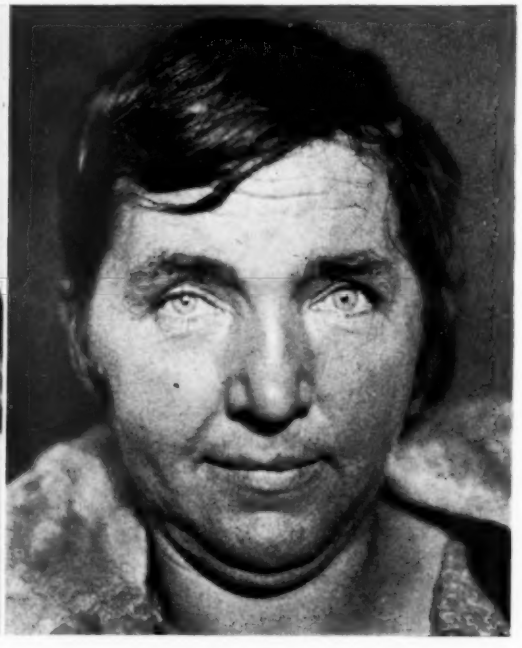


Fig. 9.

Figs. 8 and 9. Middle meningeal hemorrhage with right third nerve paralysis. In this case the pupils became equal three months after operation.

zation from the dural side was evident in sixteen hours. This organization can only obtain on the dural side, whereas, due to operation, this opening into the subdural cyst is sealed off, thus bringing about the same physical and chemical relationship

Fig. 10. Fig. 11.

Figs. 10 and 11. Case of middle meningeal hemorrhage with left oculomotor palsy. As shown in Figure 11, patient still had a dilated pupil on the left side, two and a half months after operation. This boy had bloody spinal fluid with a pressure of 600 mm. of water. He was extremely ill and would have died within a few hours. The presence of grossly bloody spinal fluid should not be a factor in excluding middle meningeal hemorrhage of benign proportions as proven in this case.

lack of blood supply, the arachnoid does not help in any way.

The thought recently expounded by Gardner and others concerning the origin and formation of large cystic contents in chronic subdural hematoma is interesting and worthwhile. It is suggested that there is transference of spinal fluid and tissue fluid into the blood clot by osmosis and diffusion, thus accounting for the progressive enlargement of the subdural pathology. But it is rather difficult to explain why this same procedure does not reobtain following evacuation of such a subdural cyst through a small opening with no effort being made at removal of the wall of the cyst. It is reasonable to assume that soon after as previously present. Of course, it is assumed that some postoperative bleeding obtains in the cyst cavity. Theoretically such a patient should return for further operative work in a short time. Some of our own cases have gone for longer than three years without any return of symptoms. What happens when the cyst wall is ruptured and the contents evacuated so that there is no return of cystic accumulation, is still a mystery.

The treatment is always surgical. It is preferable to trephine both sides of the skull even if the first opening uncovers the pathology. A single trephine opening has been found sufficient to drain the cystic contents and cure the patient. This was

done in four cases with excellent results. Subtemporal decompression on the side of the lesion was used in the remaining

Fig. 12. Chronic subdural hematoma associated with left third nerve paralysis. Complete return of extraocular movements obtained in eight weeks. The clot was found on the side of the paralyzed eye. In all cases of chronic subdural hematoma bilateral exploratory operation is in order.

cases. In this group no osteoplastic flap was performed for the care of the disease process. We usually leave a rubber drain in the subdural space which is removed in the next two days. If one treats this condition by a single trephine opening, it is advisable to insure complete evacuation of the cystic contents by postural drainage and washing out the subdural space with saline at the time of operation.

ACUTE SUBDURAL HEMORRHAGE

Acute subdural hemorrhage may be brought about by pial tears, contusion and laceration of nervous tissue. In some cases it was found to be sufficiently extensive to cover the hemispheric surfaces on both sides. In others it was more localized in its distribution and was frequently found along the region of the sylvian fissure of the brain. There is undoubtedly a good reason for such an occurrence, in that the frontotemporal region of the hemisphere is encased rather snugly in the cranial cavity with several sharp boundaries of bone extending into concavities of the brain. This is particularly true of the posterior boundary of the lesser wing of the sphenoid and its extension along the parietal bone. This bony ridge can easily cause laceration and contusion of the frontotemporal junction of the hemisphere when the brain and the bone "crowd" each other in contrecoup lesions. In eleven cases where operation disclosed the presence of subdural clots, indications for operation were quite definite and neurological findings of positive localization were present in all. There was Jacksonian epilepsy in two. Lucid interval was present in two cases. Inequality of pupils was present in five cases. Dilated pupil on the side of the lesion with paralysis or paresis of the opposite half of the body was present in five cases. Continued unconsciousness and disorientation occurred among four.

There is a pronounced difference between cases of acute subdural hemorrhage and extradural hemorrhage as concerns the general appearance of the patient. The former look much sicker, with increased temperature and definitely increased respirations; this is particularly true in the earlier stages of the condition. Of the eleven patients five died. In this group of cases, we have not included those with very small subdural clots, as frequently seen with compound fractures and depressed fractures; these do very nicely.

Cases of subdural hemorrhage constitute the more chronic types of head injury, in that if they survive the condition they remain disabled and are confined to a hospital as long as three to five months. It is interesting to note that a number of these after long stay in the hospital because of marked disorientation, in due time recover rather completely their mental function; a majority, however, have marked personality changes.

Indications for operation in this group have been the progressive character of physical findings. The appearance of a paresis a day or two after the accident, Jacksonian convulsions of increasing severity and not treatable by lumbar puncture, the presence of a dilated pupil, long periods of drowsiness and disorientation tending to

become more severe, have been some of the factors favoring intervention.

The operation of choice has been subtemporal decompression. A good sized opening is made for decompressive purposes. The use of a brain spatula of appropriate size is of great help to milk the surface of the brain and to rid it of clots. Unless great care is taken in the use of the spatula it may cause much damage to the surface of the brain.

SUBDURAL COLLECTION OF SPINAL FLUID

In some cases the most important pathologic finding at operation was the presence of a large amount of spinal fluid in the subdural space. Whether this fluid, which may be trapped in the subdural space, actually causes sufficient pressure to bring about the clinical findings, is at times questionable. In a few instances such a cause and effect relationship may be satisfactorily proved by excellent postoperative recovery; in others, the operative intervention and evacuation of the subdural collection of fluid seem to have little or no effect on the course of the clinical manifestations. That there may be associated brain damage cannot be denied, for in a great many examples of head injury a combination of several pathologic entities may obtain at the same time. Small amounts of subdural fluid found at operation are not classified under this heading. Here, an ounce or more has been obtained in all of the thirteen cases classified under this category. Indications for operation were evident in all the cases. There was a lucid interval in two followed by progressive drowsiness and disorientation. Drowsiness and marked disorientation were present in four cases. Jacksonian convulsions were present in two. Third nerve paralysis was present in one. Hemiparesis was present in four and inequality of pupils was seen in five patients.

In two patients the results were miraculous. Consciousness was recovered soon after operation. There were six deaths. Such high mortality emphasizes the correctness of the assumption that undoubtedly most of these patients have associated damage to the brain. For instance, two autopsy cases in this group showed petechial hemorrhages throughout the nervous system.

We strongly suspect that hydroma of the brain as described by Dandy is undoubtedly the same as collection of subdural spinal fluid in the majority. External hydrocephalus associated with tears in the arachnoid membrane can allow accumulation of spinal fluid in the subdural space, thus bringing about this condition. It is possible that once this situation exists a vicious circle may obtain. With accumulation of subdural fluid sufficient to cause pressure on the brain, more fluid may be pressed out of the brain and its cavities to add to the size of the subdural collection.

EDEMA OF THE BRAIN, CONTUSION OF THE BRAIN SURFACE, SUBARACHNOID HEMORRHAGE

In fifteen patients, the pathologic findings at operation were those of suffused brain surface with marked edema, and in some cases with subarachnoid hemorrhage.

The indications for operation were lucid interval in two, dilated pupil in five, hemiplegia and hemiparesis in seven, generalized convulsions in one, and Jacksonian convulsions in two cases. The greatest majority of these patients were operated on soon after entrance into the hospital, certainly within the first twenty-four hours. They were all very ill from the beginning. There were eight deaths and in the greatest number the operation was resorted to with a meager hope that a condition of benign proportions might be uncovered. These were truly undertaken for the purpose of exploration.

COMMENT ON OPERATED CASES

On glancing over the indications for operation in cases of extradural hemorrhage, acute subdural hemorrhage, subdural collection of spinal fluid, marked edema of the brain and subarachnoid

hemorrhage it is apparent that a great many had identical signs and symptoms. For instance, lucid interval was seen not only in cases of extradural hemorrhage but also among cases with different pathology. Paralysis or paresis of one-half of the body associated with dilated pupil on the opposite side was seen frequently in cases of acute subdural hemorrhage, edema and contusion of the brain and subarachnoid hemorrhage. It is therefore evident that a great many patients were operated on because of a combination of certain signs and symptoms with the hope that the uncovered pathology would be of benign proportions. I feel that intracranial exploration in cases with definite findings is justified. The results will not all be satisfactory, but were one to follow a more conservative course I am sure one would allow the exitus of many a patient with an operable condition of the intracranial contents.

SUMMARY

1. In the majority of cases of middle meningeal hemorrhage there are neurologic signs implicating the cortex on the effected side. Widely dilated pupil on the same side is seen frequently, although one case in this series had dilated pupil on the opposite side. Pupils equal in size do occur; this was true in two cases in this series.

2. Bloody spinal fluid of varying concentration does not rule out middle meningeal hemorrhage. In this series all punctured cases had bloody spinal fluid. To assume that the condition is one of subarachnoid hemorrhage because of the bloody spinal fluid is a grave mistake in these cases.

3. Lucid interval may be absent in middle meningeal hemorrhage. This was true in more than half of the cases in this series. Lucid interval may be wiped out because of (1) very rapid hemorrhage and (2) associated severe damage of the brain.

4. Lucid interval was seen not only in cases of middle meningeal hemorrhage but also in those with acute subdural hemorrhage, subdural collection of spinal fluid and edema of the brain.

5. Chronic subdural hematoma usually follows slight injury to the head, but in this series its association with severe brain injury and fracture of the skull is brought out. Seven cases had associated fracture of the skull.

6. The association of chronic subdural hematoma and relatively severe brain injury should be suspected in patients who remain unconscious, drowsy or disorientated for several weeks. Particularly if the spinal fluid pressure is high, an exploratory operation is justifiable.

7. When the question of cranial exploration arises, cases of head injury should be treated individually and if certain signs obtain operative intervention should be effected. In the presence of the proper signs exploration is justifiable even though results may not be favorable. In this clinic we are particularly impressed by a combination of all or some of the following signs as indication for exploration:

A. Dulling of the conscious state leading to unconsciousness or progressive deepening of an unconscious state already present.
B. Presence and progression of localizing signs rather than neurologic signs implicating the entire nervous system.
C. Increase in spinal fluid pressure.
D. Low pulse rate (in some cases).

REFERENCES

1. Browder, J., and Meyers, R. *Am. J. Surg.*, 31: 403, 1936.
2. Cassassa, C. B. *Proc. New York Path. Soc.*, 24: 101, 1924.
3. Cohen, I. *Arch. Neurol. & Psychiat.*, 18: 709, 1927.
4. Cushing, H., and Putnam, I. K. *J. Nerv. & Ment. Dis.*, 65: 260, 1927.
5. Dandy, W. E. Practice of Surgery. 12: 295, W. F. Prior Co.
6. Gardner, W. J. *Arch. Neurol. & Psychiat.*, 27: 847, 1932.
7. Grant, F. C. *Surg., Gynec. & Obst.*, 36: 251, 1923.
8. Gurdjian, E. S. *Arch. Neurol. & Psychiat.*, 28: 26, 1932.
9. Naffziger, H. C. *J. A. M. A.*, 82: 1751, 1924.
10. Peet, M. M. *Indust. Doctor*, 5: 7, 1927.
11. VonBergmann, E. System of Practical Surgery. 1: 206, 1904. Lea Bros. & Co.

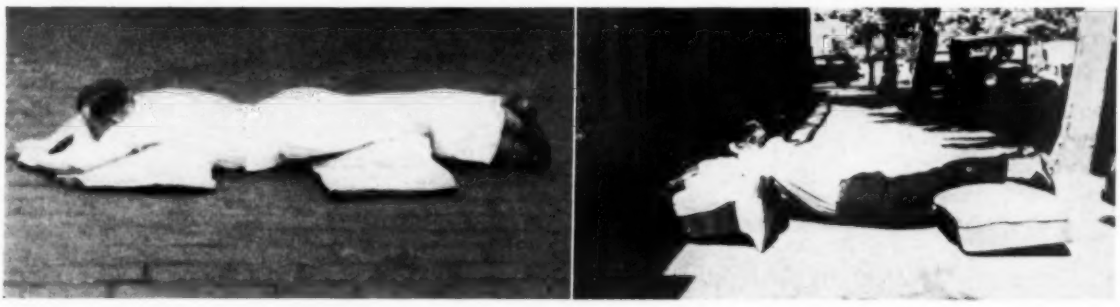
FRACTURES OF SPINAL COLUMN

A SIMPLE METHOD OF TRANSPORTATION

IRWIN E. SIRIS, M.D.

BROOKLYN, NEW YORK

THE irreparable damage that may result from the improper transportation of a patient with a fracture or dislocation of the spine merits reporting a simple procedure for transporting these patients. This method also serves as an effective aid in reduction of these injuries.



A B

FIG. 1. A, treatment at seat of injury. Position of patient. B, use of automobile seats for hyperextension of spine.

A B

FIG. 2. A, method of raising injured person. Note position of blankets and pillows. B, method of transportation on a stretcher with blanket under the head and neck and another blanket under the pelvis to hyperextend the spine.

TREATMENT AT THE SEAT OF INJURY

The public and men employed in industries should utilize the following method in handling a person who has sustained an injury to the back and who cannot move because of pain or paralysis of the legs or arms.

1. Turn the injured person so that he lies face downward.

2. Place a pillow, folded blanket, coat or the seat of an automobile under the head and shoulders and another under the thighs to hyperextend the spine. (Fig. 1.)

METHOD OF TRANSPORTATION

1. One assistant stands between the separated thighs facing the head of the injured person, and encircles his hands and forearms about the patient's thighs close to the pelvis.

2. The second assistant, facing the first, places his hands under the arm pits of the

patient and supports the patient's extended head against his body.

3. Simultaneously, both assistants gently raise the injured person, and the weight of the torso hyperextends the spine. (Fig. 2.)

The first assistant, who is holding the injured person's thighs, guides the second assistant, who walks backward or sideways as he is directed.

If the patient is to be transported with the aid of a stretcher, he is raised by the method outlined above. Then a stretcher is placed on the ground. The folded blankets, pillows, or clothes are placed on the stretcher in the respective positions where the head and shoulders and the thighs are to rest. Injuries to the spine in the region of the neck necessitate placing the blankets under the extended head. The patient is gently lowered on to the stretcher. The stretcher is then raised and placed in the ambulance. Hyperextension of the spinal column is thus maintained during transportation. (Fig. 2.)

LYMPHOSARCOMA is not infrequent in the neck, and arises either in the lymphatic glands or else in the tonsil. It may be distinguished sometimes from other malignant tumours by its very rapid growth and spread, its comparative softness, its occurrence in younger patients, and the fact that it rapidly involves many different groups of glands of the neck.
From—"The Science and Practice of Surgery" by W. H. C. Romanis and Philip H. Mitchiner, 5th Ed. (Lea & Febiger).

THE MANAGEMENT OF THE HERNIAL SAC*

RALPH WOLFE, M.D.

BROOKLYN, NEW YORK

EMBRYOLOGICALLY, the testicle, in its migration from the lumbar region into the scrotum, is preceded by a portion of the peritoneum, the processus vaginalis, which normally becomes obliterated shortly after birth. Where obliteration of the funicular process is wholly or partially incomplete, various types of inguinal hernia may develop. It can be readily understood that a persistent peritoneal sac is a potential factor in the etiology of oblique inguinal hernia. Therefore a cure for oblique inguinal hernia must concern itself primarily with the removal of the entire sac and redundant peritoneum, rather than with extensive repair of the inguinal canal.

Direct hernia, on the other hand, has no preëxisting path and is usually the result of weakness of the abdominal wall. Here redundancy of the peritoneum is not an important factor. Direct hernia is rarely observed in children and vigorous adults. It occurs most frequently in the later decades of life.

The reasons given for recurrences following the operation for inguinal hernia are extremely varied, but it is generally agreed that failure to remove the entire sac is the most common factor. Let us consider the causes of failure to remove the entire sac. Multiple sacs are the cause of recurrence more frequently than is generally supposed. In some instances an entire hernial sac may be overlooked, as in double, saddlebag or pantaloon hernia. In direct hernia, an indirect sac, which is nearly always present and should be removed, may be overlooked; and in oblique hernia, a direct sac, which may be present at times, is neglected. Both oblique and direct hernias may have bilocular sacs and unless these are found and removed, the hernia is bound to recur.

The appearance of a femoral hernia has not infrequently been observed following an operation for oblique inguinal hernia. It has been assumed in these cases that suturing the conjoined tendon to the shelving portion of the inguinal ligament has been the fault. The elevation of the inguinal ligament by this procedure in cases where relaxation of this structure is already present may have a tendency to increase the patency of the femoral canal. However, in a great number of instances, the femoral sac or peritoneal dimple probably existed at the time of the operation for the inguinal hernia, but was overlooked.

The bulge or weakness in the inguinal region which may be noted in the patient soon after operation, when he assumes the erect posture, is too often attributed to a faulty suture. More than likely an overlooked peritoneal diverticulum, funicular process, or relaxed peritoneal lining is at fault.

LaRoque states that recurrent hernia represents the incompletely removed original hernial sac or the development of an incisional rupture following breakage of suture or tissue by coughing, vomiting, or wound infection. He classifies recurrent hernias in three types:

1. Postoperative incisional ruptures following any type of abdominal incision.
2. Those in which the sac is incompletely removed.
3. Those recurrences at the lower angle of the canal having the appearance of a direct hernia.

1. Postoperative incisional hernia, whether a complication of hernioplasty or any other abdominal procedure, will be considered here only briefly. The most common factors causing its appearance are faulty suturing of the abdominal layers,

* From the surgical service of the Jewish Hospital of Brooklyn, New York.

wound infection, weak and flabby musculature of the aged and infirm, and too sudden an increase in abdominal tension. It is with

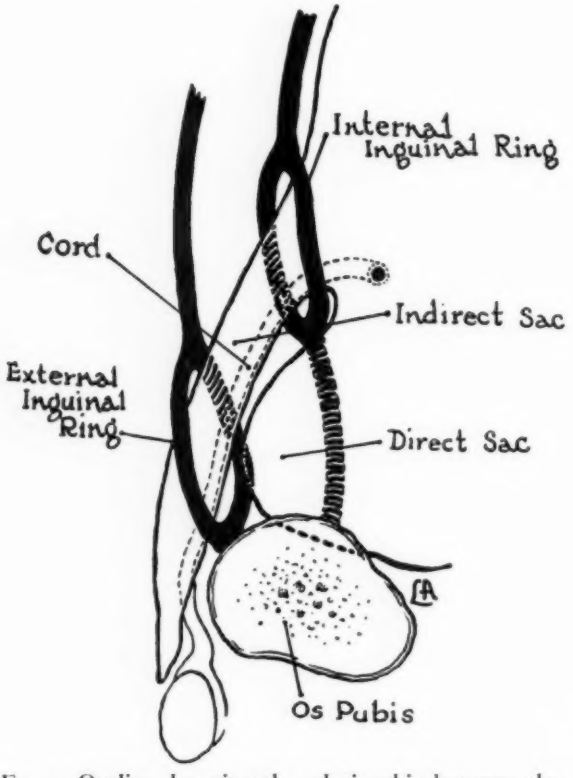


Fig. 1. Outline denoting the relationship between the internal and external rings of the inguinal canal and the direct and indirect hernial sacs.

the two latter types that we are especially concerned.

2. Failure to remove the entire hernial sac will result in a recurrence soon after operation. This may occur through careless or incomplete dissection of the oblique sac or failure to recognize the true neck of the sac. The latter condition was seen in an individual who presented a moderate sized tubular-shaped hernial sac partially closed by a small fibrotic diaphragm about one-half inch distal to the internal ring. During the operation the sac was drawn down from below the internal ring, thus leaving a funnel-shaped process or a dimple which formed the nucleus for a bulging at a later date.

3. Those so-called recurrences at the lower angle of the canal, having the appearance of a direct hernia, are not recurrences. They really indicate failure in removing the relaxed and redundant peritoneum over the urinary bladder at the time of the

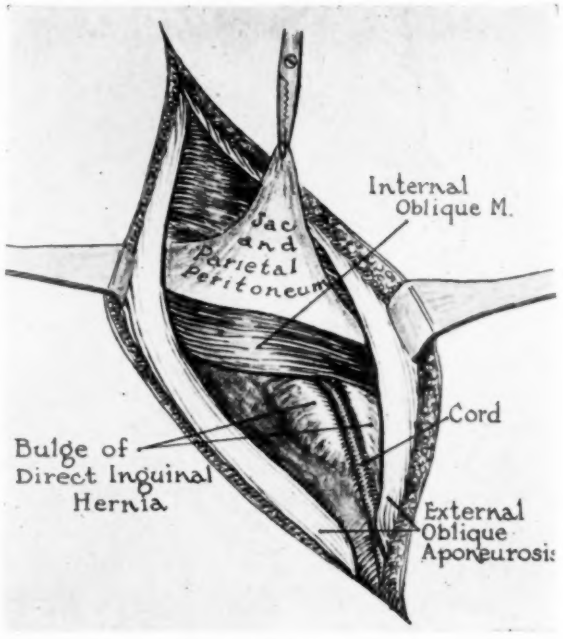


Fig. 2. The fibers of the internal oblique and the transversalis separated and the parietal peritoneum grasped above the internal ring. The bulging in Hesselbach's triangle shown.

operation for the original hernia. To obviate this difficulty and also to allow the complete removal of the sac and the redundant peritoneum, LaRoque devised his intra-abdominal operation for oblique and direct inguinal hernia. Further modifications of this principle were adopted by Banerjee and Easton.

In 1919, LaRoque described his method of attacking the problem of complete excision of the hernial sac from within the abdominal cavity. He claimed the following advantages for his procedure:

(a) High removal of the sac.
(b) Minimum of trauma to the fibers and the fascia of the cremasteric, internal oblique and the other muscles which may be utilized in wound closure.
(c) Prevention of injury to the vessels of the cord, the vas deferens, and the urinary bladder.
(d) Removal of redundant peritoneum.

Banerjee advocates the opening of the abdomen through a median subumbilical incision for the radical cure of hernia in adults. He closes the hernial opening by plicating the peritoneum about the orifice and then covering this fold of the redundant layer. By this method he not only closes the entrance to the sac, which he leaves in situ, but also irons out the dimple over the femoral ring. He states that it avoids interference with the circulation of the muscles and fascia of the inguinal region, which he believes is of prime importance in the prevention of recurrences. In celiotomy in which the coexistent hernia might be of only secondary importance, this method could be a valuable and time-saving procedure.

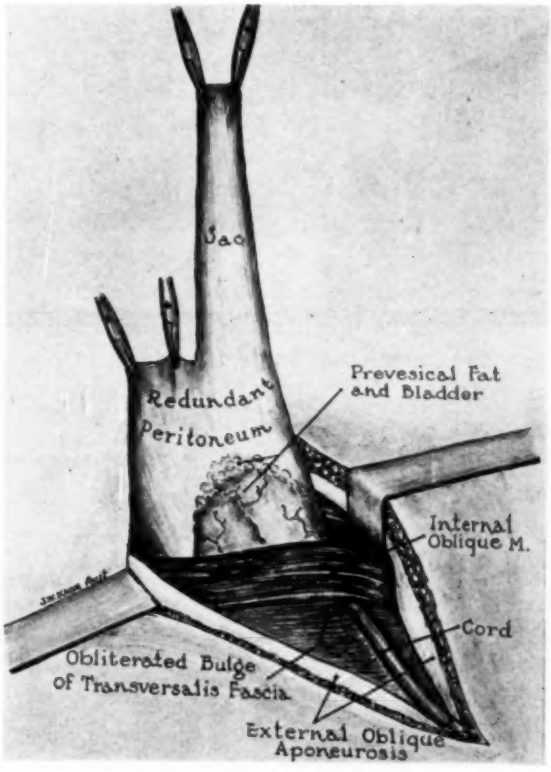


Fig. 3. The entire sac and redundant peritoneum have been withdrawn from the inguinal region. The bladder has not been separated from its attachment to the peritoneum.

Easton's operation, which combines the external approach to the sac with Banerjee's treatment of the internal orifice, by infolding of the redundant peritoneum through a curved hockey stick incision, seems unnecessarily complicated.

The following operation is described in the hope that it may offer more of the advantages and less of the drawbacks experienced with other methods. It is particularly efficacious in those patients with relaxed, flabby abdominal walls, in direct hernias, or in combined direct and indirect sacs.

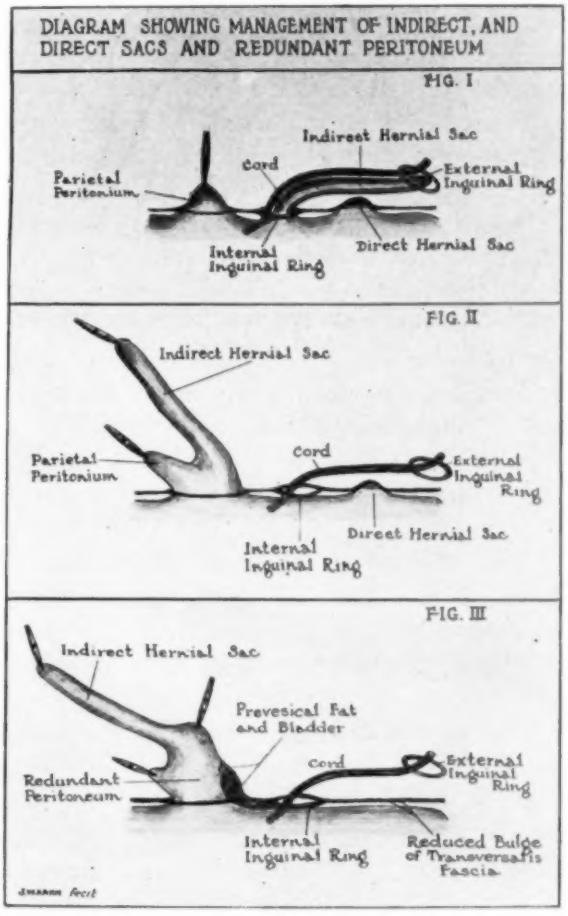


Fig. 4. Sketches I, II and III, indicating modus operandi.

The incision is the usual one from the pubic spine, 1 inch above and almost parallel to Poupart's ligament, to about 1 inch above the internal abdominal ring. The fascia of the external oblique muscle is split in the usual manner, namely in the direction of its fibers through the external ring, allowing a wide lower flap. The arched fibers of the internal oblique and the

transversalis muscles are separated by the insertion of a blunt pointed scissors through the fibers about 1 inch above the curving margin and at a higher level than the internal ring, almost in the same manner as one would approach the peritoneum in a McBurney incision.

The writer grasps the relaxed peritoneum with forceps or a hemostat in a line above the internal ring thus differing from the LaRoque technique, in which the peritoneal cavity is opened at once. (Fig. 1.) With the finger insinuated along the fold thus formed, the synechiae between the peritoneum and fascia are bluntly swept away. As the relaxed peritoneum is brought up through the wound, another hold is taken closer to its exit through the internal ring. The neck of the sac now appears. (Fig. 2.) This is grasped. The blunt handle of the scalpel is directed along the sac through the ring, releasing the sac still further. The operator now finds the plane of cleavage which permits the sac to escape easily from its attachments to the fascial tube, the vas deferens, and the spermatic plexus. With the entire sac in hand, the vas is easily swept away from the posterior wall into the retroperitoneal space without damage to its accompanying vascular structures. Further traction brings into view the yellow vascular perivesical fat and the urinary bladder. (Fig. 3.) This is brushed away from the parietal peritoneum. The sac and the redundant peritoneum are now entirely delivered into the wound and excised. The relaxation in the region of Hesselbach's triangle has disappeared. The rent in the peritoneum is closed with a running suture of catgut which also takes in a few bites of the transversalis fascia for fixation. A few plain sutures approximate the separated fibers of the internal oblique muscle. As the peritoneum is not opened until the sac is delivered into the wound, there is little danger of evisceration. The careful stripping of the peritoneum up to and beyond the deep epigastric vessels probably results in the more frequent appearance of the bladder into the wound. In large hernias in elderly or flabby individuals, the bladder is frequently drawn into the hernial wound. By this method the relaxed and redundant peritoneum can be completely removed, thus preventing recurrence. This is the important part of the procedure to obtain the desired result, a cure of the inguinal hernia.

The repair of the inguinal floor may take whatever form is appropriate to the individual case. The writer more commonly uses the repair method of Willys-Andrews or some slight modification of it. The cord is transplanted beneath the skin within its undisturbed fascial tube and above the external oblique. This permits a more perfect closure of the two aponeurotic flaps of the external oblique and almost complete obliteration of the external ring. The fascial tube prevents adhesion of the vas to the scar.

Forty-one patients were thus treated for inguinal hernia. Twenty cases could not be reached for follow-up. The results in the remaining twenty-one cases, all male, are shown in Table 1.

Table 1

Hospital No.	Age	Hernia	Type	Procedure	Result	Years
147630	23	left	direct	author's	cured	5
147936	21	left	indirect	author's	cured	5
149317	48	right	pantaloon	author's	cured	5
		left	direct	author's	recurrence	½
153092	65	right	indirect	author's	cured	5
153225	36	left	indirect	author's	cured	5
154237	51	right	pantaloon	author's	cured	5
158167	60	right	pantaloon	author's	cured	4
		left	pantaloon	author's	cured	4
161203	55	right	indirect	Bassini	recurrence	1
		left	pantaloon	author's	cured	4
165512	62	left	indirect	author's	cured	4
168102	46	right	pantaloon	author's	cured	3
		left	indirect	Bassini	recurrence	⅔
177873	65	left	direct, indirect & femoral	author's	cured	2
178655	37	right	pantaloon	author's	cured	2
		left	pantaloon	author's	cured	2
181289	37	right	indirect	author's	cured	2
182386	45	right	indirect	author's	cured	2
182662	25	right	pantaloon	author's	cured	2
189932	34	right	direct	author's	cured	1
189377	27	right	pantaloon	author's	cured	1
189412	48	left	direct & femoral	author's	cured	1
191671	47	right	pantaloon	author's	cured	1
192701	36	left	pantaloon	author's	cured	1
194339	47	left	direct	author's	cured	1

Twenty-six operations were performed in this group as follows:

Indirect	8
Direct	3
Pantaloon (combined direct and indirect sacs)	13
Recurrent direct	2
Femoral	2

Femoral hernia appeared twice as a complication of the inguinal variety and was therefore repaired through the inguinal incision. Acute strangulation and an undescended testicle appeared once in this series.

Five patients were subjected to bilateral hernioplasties and in this group there were three recurrences. In one instance the recurrence followed the author's technique and in two cases the classical Bassini procedure. Apparently this would indicate that the simultaneous surgical repair of bilateral hernias tends to increase the incidence of recurrence.

Postoperative Course. Temperature reaction ranged from 99 to 102.8 degrees. Temperatures over 100 degrees were seldom seen after the third day. Hospital stay averaged fifteen days; for bilateral hernias, seventeen days, and for unilateral hernias, twelve days. One patient with a recurrent direct hernia on whom an extensive iliotibial fascial transplant was performed, stayed twenty-three days.

COMMENT

Sliding and other anomalous forms of hernia may be readily recognized. The incarcerated and strangulated varieties may be inspected from within the abdominal cavity and appropriate measures applied. Any form of repair may be utilized. Occasionally one has some technical difficulties with a very thin peritoneum because of tearing. However, with care this too can be handled with the usual routine procedure.

This technique is especially adapted to recurrent cases. In such instances the redundant peritoneum or sac may be removed without disrupting the entire sutured canal. There is little or no trauma to the cord and its vascular components. The infundibuliform fascial tube is left intact about the cord. The bladder may be easily recognized and steps taken to prevent accidental injury.

SUMMARY

A method for the management of the hernial sac and redundant peritoneum is described. It presents the following advantages:

1. High removal of the sac and redundant peritoneum.
2. Inspection of the abdominal cavity if necessary.
3. Exposure without trauma to the vas deferens and the bladder.
4. Adequate repair of the inguinal canal.

I am indebted to Dr. Jacob W. Kahn for the accompanying illustrations.

REFERENCES

1. Andrews, E. W. *Chicago Med. Rec.*, 9: 67, 1895.
2. Banerjee, P. *Surg., Gynec. & Obst.*, 54: 706, 1932.
3. Easton, E. R. *J. A. M. A.*, 100: 1741, 1933.
4. LaRoque, G. P. *Surg., Gynec. & Obst.*, 24: 507, 1919.
5. Watson, L. F. Hernia. St. Louis, C. V. Mosby Co., 1924.

RECONSTRUCTION OF INTERNAL RING WITH ATTACHED FLAPS OF APONEUROTIC FASCIA IN INDIRECT INGUINAL HERNIA

PRELIMINARY NOTE

M. BENMOSCHE, M.D.

Senior Clinical Surgeon, Mount Sinai Hospital

NEW YORK CITY

THE mechanism of the production and development of indirect inguinal hernia is still an unsolved problem, while the technique of hernioplasty has been so well covered as to leave apparently little room for improvement.

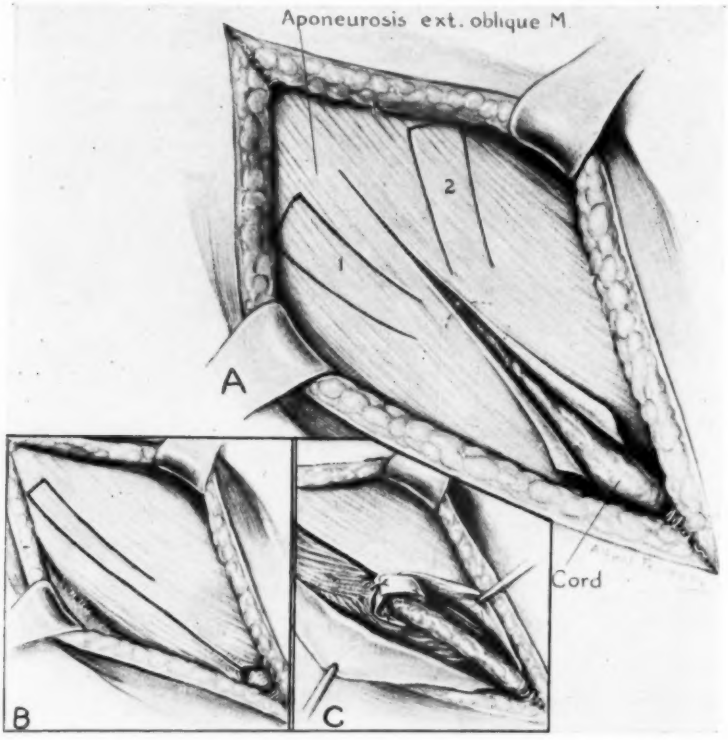


FIG. 1. A, wide exposure of aponeurosis of ext. oblique. Pedicled flaps outlined. Aponeurosis centrally divided. B, single flap outlined when flap proves impracticable. C, disposition of single flap around internal ring.

Hernia makes an interesting chapter in the history of progressive surgery. Joseph Pancoast,[1] in his "Treatise of Operative Surgery," published in 1846, makes several interesting observations in his chapter on the Radical Cure of Hernia: Mention is first made of operations which "have for a long time been abandoned, viz.:

"1. Castration.

"2. Cauterization upon the surface of the skin, or upon the neck of the sac after the skin has been laid open so as to expose the cavity.

"3. Ligation of the neck of sac with a gold thread, known as the golden stitch.

"4. The Royal Stitch, which consisted in sewing up the neck of the sac, and excising

all the body of the sac below the line of suture.

"5. The Spanish process, in which the sac was laid open for the purpose of pushing the testicle into the cavity of the abdomen, and closing the neck with the golden stitch.

"6. Reduction of the hernial sac entire after having previously dissected it up from its attachments."

A method of "Acupuncturation" is then described: "It consists in making from the surface of the skin one or two rows of punctures with a common acupuncture or large sewing needle across the neck of the sac immediately below the orifice of the external ring." This, it appears, was intended to produce an "irritation" and a final "obliteration" of the sac. Very soon after it was found that there was a "more positive means of exciting inflammation by the instillation of a few drops of some highly stimulating fluid into the cavity of the sac." Then follows a lengthy description of the process of curing inguinal hernia by "injection . . . as employed by the author."

Bassini,[2] in 1883, attempted the radical cure of indirect inguinal hernia by using the stump of the sac as a stopper to the internal ring. One year later he devised the classic operation which bears his name and which is most commonly performed today, with or without various modifications, as the operation of choice in this type of hernia. In 1890, he published his results in 262 hernias operated upon by him, in which the present Bassini operation was employed.

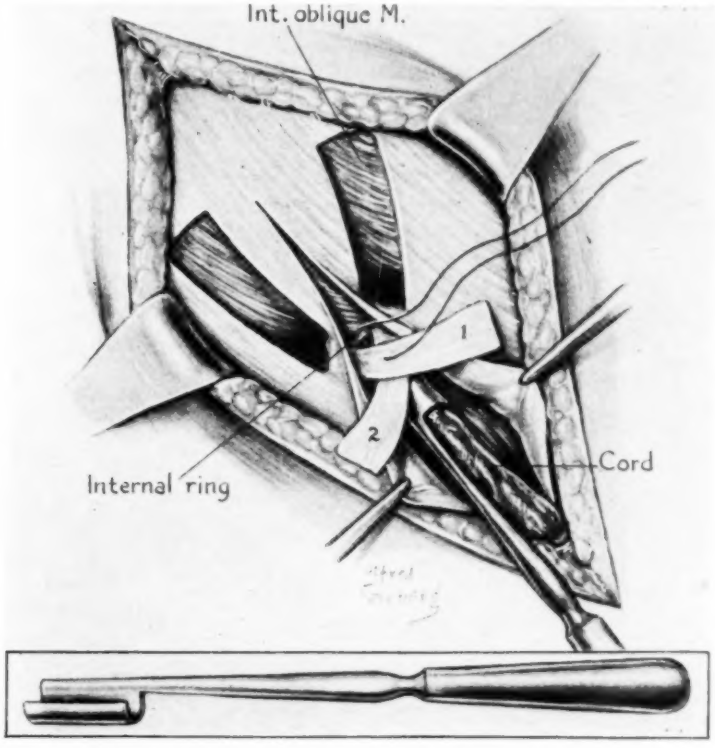


Fig. 2. Pedicled flaps inverted, crossed over each other and tied, with cord retractor in position protecting cord. Insert shows cord retractor.

Watson[3] describes the obliquity of the inguinal canal as being a natural obstacle to hernia, because an increase in intra-abdominal tension forces the inguinal walls more firmly together. It appears that Bassini's original thought was primarily to "make it unnecessary for the patient to wear a truss after operation." Obviously what he had in mind was that the mechanical effect of wearing a truss could more

permanently be reproduced by his operation of reconstructing the canal, and making a new bed for the cord by suturing the conjoined tendon to the "posterior margin of Poupart's ligament."

I am inclined to discount the importance which has been given to the pathologic changes of direction of the canal, which apparently take place only in indirect inguinal hernia. Regardless of change of direction, or the thin and weakened structures which may constitute the immediate environs of an indirect inguinal hernia, it is inevitably through an imperfect internal ring that the sac and its contents must gain entrance to the canal. The recent revival of methods of injecting the rim of the ring for the purpose of constricting it by cicatrization, is an indication that the trend of thought is toward the basic importance which the ring plays in the etiology of this type of hernia, although the procedure itself, for obvious reasons, should be severely condemned.

The physiologic adaptability of fresh sutures of fascia lata in hernia repair has been definitely established. It has also been shown that attached tissue transplants fare better, on the whole, than when they are detached.

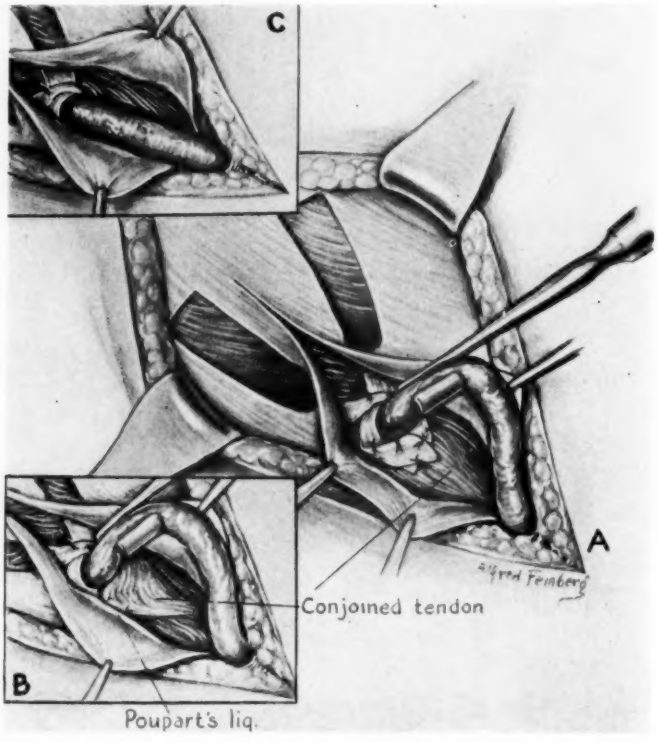


Fig. 3. A, pedicled flaps sutured around the margin of ring, overlapping at four points of circumference. B, upper fibers of conjoined tendon sutured to Poupart's ligament. C, cord placed following reconstruction of ring.

With this in mind, the following operative procedure has been devised, and its practicability demonstrated on the cadaver:

Dissection exposes a wide expanse of the aponeurosis of the external oblique through an incision parallel to Poupart's ligament and approximately 3 cm. internal to it, extending from a point about 4 or 5 cm. below the anterior superior spine of the ilium down to about 2 cm. from the pubic spine. The dotted lines in Figure 1 indicate from which part of the aponeurosis the flaps may be secured. Insert B shows an alternative method of obtaining a single fascial flap and Insert C illustrates its final disposition. The tensile strength of flaps parallel or at right angle to the direction of

the aponeurotic fibers is approximately identical.

Exposure of the cord, isolation of the sac, identification of contents and their reduction with high ligation of sac are carried out in the usual manner of the Bassini operation.

Figure 2 illustrates the pedicled formation of two aponeurotic flaps as to required length and width. This is the method of choice. The use of one long strip of fascia, as shown in Insert B of Figure 1, may be employed as an alternative method. The flaps are inverted and cross each other at the upper margin of the ring to which they are sutured before proceeding further. Insert C (Fig. 1) shows method of inverting the single flap.

The cord-shield and retractor which I have specially devised for this procedure, in position as illustrated, protects the cord in the process of suturing the flaps to the ring in the several positions around the cord.

The flaps are sutured to the lateral margins of the ring and overlap themselves at four points of the circumference. (Fig. 3.) Insert B illustrates the manner in which the upper fibers of the conjoined tendon sutured to the overshelving portion of Poupart's ligament may reinforce the lower portion of the ring without actually reconstructing the entire canal. Insert C shows the cord in final position. The aponeurotic repair is completed, as shown in Figure 4.

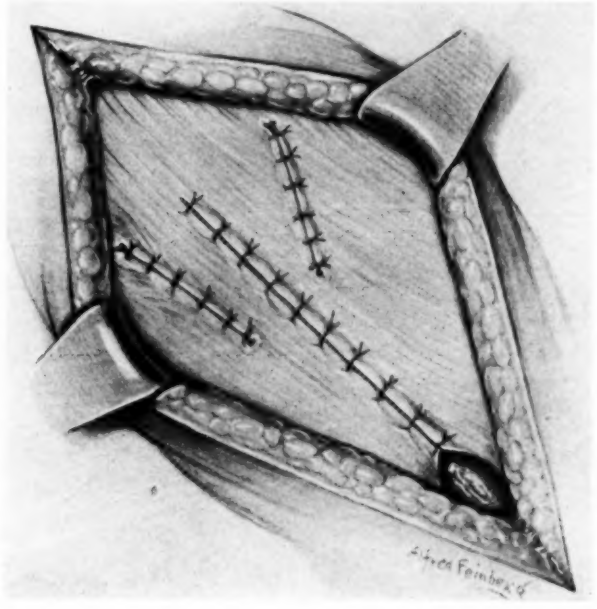


Fig. 4. Final repair of operative defects of aponeurosis of external oblique.

SUMMARY

1. The basic importance of the imperfect internal ring in the production of indirect inguinal hernia is stressed.

2. A brief history of methods of dealing with indirect inguinal hernia, leading to the present day conception of treatment of hernia by injection, is given.

3. A method of reconstructing the internal ring with attached flaps of fascia from the aponeurosis of the external oblique is shown to be mechanically practical.

REFERENCES

Pancoast, Joseph. Treatise of Operative Surgery, Phila., 1846. Carey and Hart.

Bassini, Eduard. *Arcb. f. klin. Cbir.*, 40: 429, 1890.

Watson, Leigh F. Hernia. St. Louis, 1924. C. V. Mosby Co.

THE SQUARE KNOT: RATIONALE OF TECHNIQUE*

A ONE-HANDED "THUMB-ROLL" MANEUVER

EMANUEL GOLDBERGER, M.D.

NEW YORK CITY

INTRODUCTION

THE square knot consists essentially of loop within a loop. (Fig. 1.) Concerning its production, Livingston[1] in a masterly paper, demonstrated that it must depend, fundamentally and physiologically, on but two types of maneuvers, either:

(a) "the maneuver of ambidexterity," in which identical movements of first one, then the other hand, produce the square knot;

(b) "the maneuver of bimotility," in which a square knot is obtained when the two component ties are made by opposite motions of the same hand.

Thus a criterion may be established, namely: that the value of a knot-tying technique should be measured by the relative movements of the hands and fingers, rather than the varied relations of the cords (which in the final analysis are dependent on the former).

In other words, that procedure is best which (a) entails the least movement of the fingers and hands; (b) obviates or reduces to a minimum cumbersome motions, as crossing of the hands, and especially supination, and extension of the fingers, the awkwardness of which has been amply demonstrated;[1] (c) insures control of the tying procedure during all stages of the maneuver; (d) avoids displacement of the first simple knot (Fig. 2), which would result in a loose and insecure tie; (e) is fastest.

If the needs of the surgeon be analyzed, it becomes apparent that no one procedure, be it bimanual, one-handed, or instrumental, offers itself as the ultimate in perfection for all occasions. For the usual situation, the bimanual technique serves well. With respect to variations in this maneuver[1–8] discussion here is out of place, though the criteria presented above apply just as well; and the same general rules can be applied to the instrumental techniques[1,9–14] which are of distinct value when a "no-hand-touch" asepsis is required, etc. Also, with this in mind, it should be an easy task to ascertain the value of those methods primarily consisting of tricks[15] and flashy maneuvers with but little practical advantage.

The scope, then, of this paper lies with the technique of the one-handed knot, which has its chief value when one of the surgeon's hands is occupied with a long suture or needle-holder, a condition that not infrequently occurs.

I desire to present first the maneuver I employed—one as effective as it is simple—and then to compare it, in the light of the criteria presented above, with the one-handed procedures which have had the merit of publication.

TECHNIQUE

General Directions (of value no matter what manual procedure is employed). Mention should be made of the fact that there is no so-called one-hand maneuver which utilizes one hand exclusively; the second is absolutely necessary in tightening the knot made by the one hand, if displacement of the first knot or a loose tie is to be avoided.

In the case of a ligature, the square knot should always be tied to effect the long axis of the knot (Fig. 1) in parallel relation to the sagittal plane of the surgeon's body; or the long axis of the wound to be sutured should be in relation to the frontal plane

* From the Department of Surgery, Trinity Hospital, Brooklyn, New York, Dr. Julius B. Boehm, Director.

of the body. For this, the surgeon may find it necessary to twist his trunk slightly.[1] The value of this is that it obviates crossing of the hands when the knot is tied, the hands moving rather toward and away from the body.

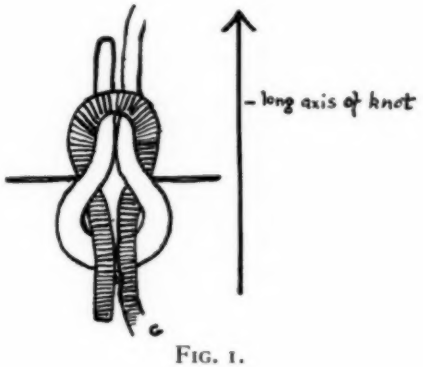


Fig. 1.

The One-Hand Thumb-Rolling Maneuver. Only the thumb and index finger are employed.

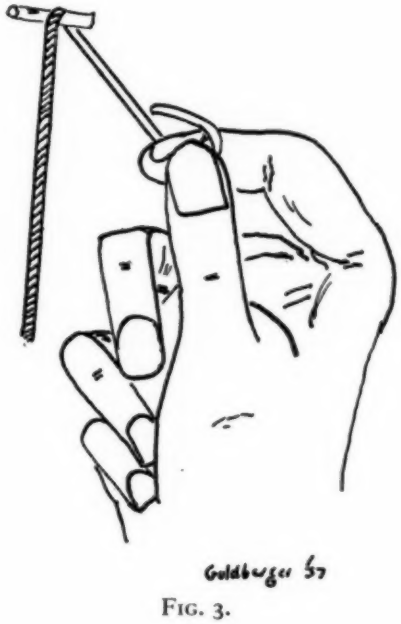


Fig. 3.

1. Grasp the short end with the thumb and index finger of the right hand. (Fig. 3.)

2. Bring the sides of the long and short ends together. (Fig. 4.)

3. Slide the thumb toward the tip of the index finger, and slide the index finger similarly, causing the short end to roll over and around the long end in a counter-clockwise direction, until a complete rotation has been effected.

4. Continuing to hold the short end between the thumb and index finger, pull the right hand away from the body, and the left toward the body slightly. This completes the first tie, a simple knot. (Fig. 5a.)

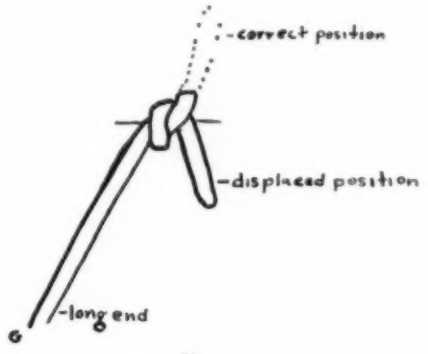


Fig. 2.

5. Twirl the index finger, with the aid of the thumb, around the short end, reversing the direction of the free end. (Figs. 5b, c.) This serves to reverse the first maneuver.

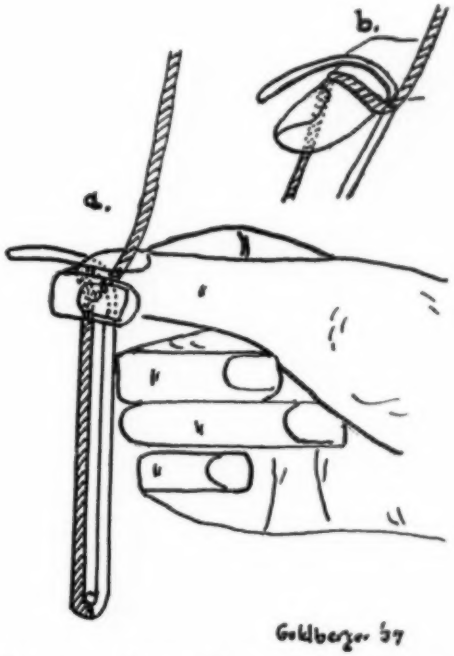


Fig. 4. A, lateral view. B, looking from above on index finger. Thumb has been raised.

6. Bring the sides together, as in step 2. (Fig. 6.)

7. Again, roll the short end counterclockwise over and around the long end.

8. Tighten the second tie, pulling with the right hand toward the body, and with the left, away. This completes the square knot.

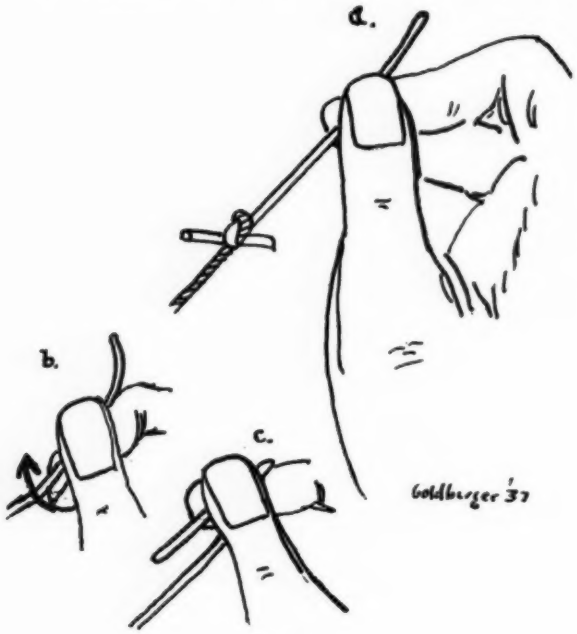


Fig. 5.

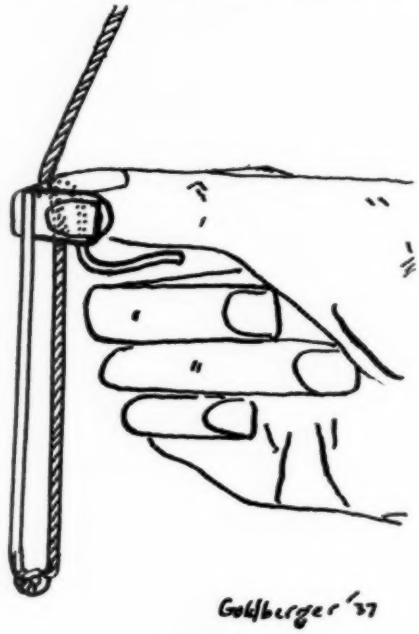


Fig. 6.

DISCUSSION

As has been said before, the efficacy of a knot-tying technique can be judged by the number of hand and finger movements necessary; the number of times the hand must be supinated and pronated; how many times there are changes in the suture (or ligature) hand relationship, not related directly to the tie; the degree of control obtained; and the speed. (Table 1.) With respect to speed, this is a factor so dependent on one's familiarity with the technique used that its inclusion as an index of comparison is not warranted.

Table 1
ONE-HANDED KNOT-TYING MANEUVERS

Technique	No. Finger and Hand Movements	No. Times Supination and Pronation	No. Changes of Cord-Hand Relationship	Displacement of First Knot
Author's	8	0	1	no
Livingston[1]	8	3	1	no
Meynen[16]	8	3	1	no
Sullivan[17]	not a square knot but "surgical."			
Monks*[18]	10	3	3	yes
Richardson[3]	9	1	2	a badly tied knot
Kubo[19]	not a square knot, but "surgical."			

* This technique necessitates crossing of the hands twice.

The same standards were employed in calculating the number of finger and hand movements (or steps) etc. in the procedure described above, as for the others noted in Table 1.

On the basis of the values obtained above, I can no more than conclude that the procedure I employ, compared to those I was able to find mention of, is the simplest. The effectiveness, of course, of any method which follows the general principles outlined above must be the same. That is to say, a square knot is a square knot whether it was tied in two or twenty seconds, or with one hand or four instruments.

SUMMARY

1. The general principles of tying the square knot have been enunciated.

2. Indications for various techniques have been described.

3. A new and simple procedure for the so-called one-handed square knot has been presented.

4. Comparison has been made between this and what other methods had been found in the literature.

REFERENCES

1. LIVINGSTON, E. M. Study of the square knot. *Am. J. Surg.*, 6: 121 (Feb.) 1929.
2. BRYANT, J. D. Operative Surgery. 1: 69. New York, 1901. D. Appleton & Co.
3. BICKMAN, W. S. Operative Surgery. 1: 238–244. Phila., 1924. W. B. Saunders Co.
4. BULININ, I. Zur Frage der Technik des Schützens von chirurgischen Knoten. *Arch. f. klin. Chir.*, 179: 526, 1934.
5. SULLIVAN, W. J. Technic of a knot. *J. A. M. A.*, 80: 180 (Jan.) 1923.
6. VON MEZO, B. Ein einfache und schnelle Methode zur Knüpfung des chirurgischen Knotens. *Zentalbl. f. Chir.*, 48: 886 (June) 1921.
7. ZIMCHES, I. L. Zur Technik des chirurgischen Knoten. *Zentralbl. f. Chir.*, 56: 602 (March) 1929.
8. ELIASON, E. L. Method of tying a flat knot. *J. A. M. A.*, 76: 268, 1921.
9. GRANT, A. R. The tying of a surgical knot. *Ann. Surg.*, 67: 439 (April) 1918.
10. FARR, R. E. Three forceps and four forceps knot. *Surg., Gynec. & Obst.*, 31: 408 (Oct.) 1920.
11. FORRESTER, C. R. G. Imperative Traumatic Surgery. New York, 1929. Paul B. Hoeber. Page 403.
12. WEIGL, F. Instrumentelle Fadenknotung. *Med. Klin.*, 20: 1113 (Aug.) 1924.
13. FECHER, K. Zur instrumentelle Knotenbildung. *München. med. Wchnschr.*, 69: 1217 (Aug.) 1922.
14. VARELA, V. D. Instrumentelle Knotenbildung. *Zentralbl. f. Chir.*, 47: 266 (March) 1920.
15. HAVENS, F. Z. Trick in tying a knot. *Surg. Clin. North America*, 15: 1229 (Oct.) 1935.
16. MEYNEN, F. G. Method of tying a square knot. *J. A. M. A.*, 85: 579 (Aug.) 1925.
17. SULLIVAN, W. J. One-handed surgical knot. *Am. J. Surg.*, 39: 8 (Jan.) 1925.
18. MONKS, G. H. Tying the knots of ligatures and sutures with one hand. *Ann. Surg.*, 56: 780, 1912.
19. KUBO, I. Ein Methode des chirurgischen Knotens. *Arch. f. Obren., Nasen. u. Keblkopfb.*, 122: 178 (Aug.) 1929.

SIMPLIFICATION OF DATA ON THE SERIOUSLY SICK SURGICAL PATIENT

CARL BAUMEISTER, B.S., M.D. AND C. W. HARGENS, M.D., F.A.C.S.

COUNCIL BLUFFS, IOWA

THE seriously sick surgical patient presents many problems. Not one of the least of these is a satisfactory method of recording the ever changing clinical picture of the patient.

Date	Fluid Intake & Parenteral c.c. in 12 Hours				Output		Gas Rectal BM	Duodenal Gas	Urinalysis				B.P.	Remarks
									Su	Al	Ac	Ch		
	Retain Ora	Procto	SubQ	Intrav	Emesis	Urine		Asp c.c.						

FIG. 1.

The following method has been incorporated on the surgical service at the Sisters Hospital in Hot Springs, South Dakota:

1. The temperature, pulse and respirations are recorded as usual on the standard sheet.

2. The standing orders are copied onto a separate sheet every twenty-four hours by the night shift. In this way each morning only the live orders are present on the sheet.

3. A third sheet has been originated. (Fig. 1.) All columns are filled in each twelve hours, a double line being drawn between days. Some of the columns are self explanatory. Under fluid intake are included the four possible routes. In many patients with a Levine tube with the Wangensteen suction attached there is nevertheless some fluid retained orally. While most of these patients get fluid intravenously, columns are left for recording of the subcutaneous and proctoclysis fluids. As a rule 1000 c.c. of 5 per cent glucose in saline is given at the start of the day, the remaining fluid being 5 per cent glucose in distilled water. On long continued cases we prefer the intravenous cannula.

Under fluid output we usually attempt to have 1500 c.c. of urine daily. A space is left for emesis, its character and amount usually being recorded.

Under gas we have left space for the amounts of both rectal and duodenal gas. A Wangensteen apparatus is often attached to a rectal tube in cases with distention, particularly that of the lower bowel, in addition to the inlying duodenal tube with suction. B.M. can be recorded under the rectal gas column.

Urinalysis is routine every twelve hours in these cases. Sugar, albumin, acetone and hydrogen ion concentration are checked. We very rarely use insulin in our intravenous glucose solution but are on a constant watch for a spill in the urine which indicates its use.

Blood pressure has a column to itself. A check on this every six hours (two B.P. to the column) warns us of early peripheral vascular or cardiac failure which is so important, particularly in ileus and respiratory complications.

Under Remarks is left a space wherein other essential data can be placed. These are chiefly laboratory data or unusual clinical facts noted by the nursing staff. Under Laboratory often comes total white counts, less often blood sugars and non-protein nitrogens.

The use of these three sheets in the manner mentioned has greatly simplified and clarified in our minds the care of the seriously sick surgical patient.

Patients with enormous effusions [acute empyema thoracis] causing respiratory distress are best treated by aspiration. The amount of fluid withdrawn should be determined by the relief obtained.
From—"Pediatric Surgery" by Edward C. Brenner (Lea & Febiger).

MONOETHANOLAMINE OLEATE*

A NEW CHEMICAL FOR THE OBLITERATION OF VARICOSE VEINS

NATHANIEL E. MEYER, M.D.

NEW YORK CITY

THE use of sodium morrhuate for the injection of varicose veins has been widely adopted because of its superiority over most of the other agents.

Its advantages were listed as follows:

1. It is practically painless.

2. There is diminished likelihood of slough when small amounts are infiltrated during an injection.

3. Its efficacy is at least comparable to the other agents in producing a firm thrombus.

However, there have been appearing in the literature repeated reports of allergic manifestations following the use of sodium morrhuate. These manifestations vary from mild erythema to severe generalized urticaria and even anaphylactic shock. There are probably many more of these cases that never reach the literature. The author has observed numerous such reactions to morrhuate, especially of the urticarial variety. Conversation with other physicians has revealed that these reactions are not uncommon.

Upon considering the composition of sodium morrhuate, the reason for this high incidence of allergic reaction becomes obvious. Its exact composition, even in the purified form, is unknown. It is virtually impossible to remove all the fish proteins which are present, in small amounts to be sure, but sufficient to cause hypersensitivity in allergic subjects.

Monoethanolamine oleate has been synthesized in the chemical laboratory and is of known composition. As the name implies, it is a soap of monoethanolamine and oleic acid. The structural formula is $CH_3(CH_2)_7CH{=}CH(CH_2)_7CO{-}NH{-}CH_2(CH_2)OH$. The compound is a yellowish viscid liquid, soluble in water. Its viscosity in a 5 per cent solution is somewhat less than sodium morrhuate. Monoethanolamine oleate has been tested in the biologic laboratory. The minimum lethal dose intravenously in rabbits is 130 mg. per kilo, as compared to 100 to 125 mg. per kilo in sodium morrhuate.

In the chemical and biologic laboratory the compound was found to be satisfactory and at least as safe as the older solutions. There remained the proof of clinical trial.

Forty-three consecutive cases were injected with the solution. Of these fifteen were males and twenty-eight females. A total of 345 injections were given. The technique used was virtually the same as with sodium morrhuate. Single injections were given once a week and the amount of thrombosis noted the following week. The patients were questioned for any possible reactions. The initial injection was usually 1 to 2 c.c. The thrombosis was noted the following week and the amount of subsequent injection regulated as required by the individual patient and the size of the vein. No tourniquets were used except in cases where there was a failure of or insufficient thrombosis. For statistical purposes, the resulting thromboses were divided into the following classes:

Class 0: Failure of thrombosis.
Class 1: Thrombosis up to one-half inch.
Class 2: From one-half inch to 2 inches.
Class 3: Over 2 inches or with chemical phlebitis, but with no tendency to break down.
Class 4: Ascending chemical phlebitis.

Thromboses in Class 2 were considered optimum. While Classes 3 and 4 produced good end-results, we felt that they were

* From the Out-Patient Department of Lincoln Hospital, New York City.

undesirable because discomfort often prevented the patient from doing his usual work, so that treatment could no longer be considered ambulatory.

The thromboses produced were as follows:

Unrecorded:	5 injections
Class 0:	11 injections
Class 1:	27 injections (Note: Most of these resulted from the small amount used in the initial injection.)
Class 2:	284 injections
Class 3:	16 injections
Class 4:	2 injections

There were no sloughs in spite of the fact that on numerous occasions small amounts were infiltrated. There was no evidence of allergic reaction in any of the patients.

The volume of solution injected varied from 1 to 5 c.c., the usual amount required being from 2 to 3 c.c. Of the 345 injections, 287 were from 2 to 3 c.c., and 203 from 2.5 to 3 c.c.

At one time we ran short of the oleate, and substituted sodium morrhuate temporarily in many of the cases. Following the morrhuate injections there resulted two cases of urticaria. One patient refused further treatment. In the other case treatment was continued with the oleate, and the urticarial symptoms subsided. After two weeks a small amount of sodium morrhuate was again injected with return of the urticaria.

SUMMARY

A clinical trial of a new chemical, monoethanolamine oleate, was made on forty-three cases; a total of 345 injections were given. In every case a good thrombus could be produced either at first or subsequently on injection. There were no cases of slough or any evidence of allergic reaction.

CONCLUSION

Monoethanolamine oleate is a satisfactory substitute for sodium morrhuate. It is comparable to the morrhuate in its lack of pain, its efficacy in production of thrombosis and prevention of slough. It is superior to morrhuate because it is of known standard composition, it is more stable in solution, and has less tendency to produce allergic reactions.

The monoethanolamine oleate for this study was supplied through the courtesy of the Abbott Laboratories.

REFERENCES

Biegeleisen, J. The evaluation of sodium morrhuate therapy in varicose veins. *Surg., Gynec. & Obst.*, 57: 696–700, 1934.

Frohman, I. G., and Brand, G. D. Complications in injections of varicose veins. *J. A. M. A.*, 103: 1088 (Oct. 6) 1934; 103: 1794 (Dec. 8) 1934.

Keitzer, W. A. *J. Michigan Med. Soc.*, 35: 168–169 (March) 1936.

Lewis, K. M. Anaphylactic shock due to sodium morrhuate. *J. A. M. A.*, 107: 1298 (Oct. 17) 1936.

McAusland, S. *Brit. M. J.*, 1: 430 (Mar. 11) 1933.

Wells, C. A. *Brit. M. J.*, 1: 674 (April 15) 1933.

Zimmerman, L. M. *J. A. M. A.*, 102: 1216–7 (April 14) 1934.

CASE REPORTS

PERIRENAL TUMORS CAUSING RENAL DISTORTION

REPORT OF A CASE OF PERIRENAL FIBROMA

JOSEPH A. LAZARUS, M.D.

NEW YORK CITY

ANY retroperitoneal tumor in the vicinity of the renal fossa or ureter may cause sufficient distortion of the kidney or ureter to give rise to symptoms referable to the genitourinary tract. Distortion may be caused: (1) by direct pressure of the tumor upon the kidney or ureter; or (2) by retraction, especially in cases of enlarged retroperitoneal tuberculous lymph nodes resulting in (a) rotation of the kidney upon its vertical or horizontal axis, (b) compression or angulation of the renal pelvis, and (c) compression or dislocation of the ureter. In fact, Andrews,[1] reporting on twenty-eight cases of retroperitoneal sarcoma, noted six patients (21 per cent) complaining of increased urinary frequency, and one of dysuria and hematuria.

To enter into a detailed discussion of the entire subject of retroperitoneal tumors would lead one too far astray from the purpose of this communication. Suffice it to say, however, that such neoplasms are not rare. Gobell,[4] in 1901, collected 101 cases of retroperitoneal tumors, of which thirty-two were malignant, forty-three solid benign and twenty-six cystic benign. Andrews, in 1923, was able to collect 142 cases of primary retroperitoneal sarcoma including twenty-eight cases from the Mayo Clinic, while Magoun[5] reported seventy-three cases of general retroperitoneal tumors of all types from January 1907 to September 1919, collected from the records of the Mayo Clinic. Of this number fifty-three were operated on, disclosing twenty-nine malignant tumors and eighteen benign. Six were undetermined.

Steele,[8] in 1900, collected sixty-one cases of retroperitoneal sarcomata, of which number forty-four cases were subjected to microscopic examination, disclosing eleven round-cell sarcomata, twelve spindle-cell, seven spindle and round-cell sarcomata, five lymphosarcomata, eight myxosarcomata and one endothelioma. Mauclair[6] classified such tumors in the following groups: pure lipoma; pure fibroma; myxoma; chondroma; myoma; neuroma; sarcoma; lymphadenoma; and all varieties of cysts.

The sources of origin of retroperitoneal tumors are: (1) the retroperitoneal lymph nodes (64 per cent of Steele's series); (2) the connective tissue around the spine, the sheath of vessels, the iliac region, the pelvis and around the kidney; and (3) the endothelium of lymphatic radicles around the kidney. In Steele's series 36 per cent arose from connective tissue of the retroperitoneal spaces and the sheaths of the great vessels. This same author gave the site of origin of the tumor as the lumbar region in 57 per cent of the cases and the central portion of the abdomen about the attachment of the mesentery in 41 per cent of the cases. In the lateral tumors Steele found the right side was much more often affected than the left, and in most of these lateral tumors there was some involvement of the corresponding kidney. The tumor was either adherent to or surrounded the kidney.

The largest malignant tumor on record was a myxo-fibro-chondrosarcoma, weighing 34 pounds, reported by Bull.[2] Of the benign tumors the lipomata grow the largest. Most of the malignant tumors, when found at operation or at post-mortem, were large. Twenty-three of the twenty-eight tumors of the Mayo series reported by Andrews were in males. In only one case in this series were multiple tumors found. Unlike the findings in Steele's series, thirteen of the growths were found on the left side and nine on the right; three were in the pelvis; two were noted as being centrally located; and one extended across both sides of the abdomen.

Fig. 1. Flat film showing the relationship between the tumor and the right kidney.

Fig. 2. Intravenous pyelogram showing the folding distortion of the right pelvis and the displacement of the upper portion of the right ureter.

Symptoms. Symptoms are naturally dependent upon the size of the tumor and its proximity to and involvement of neighboring structures, so that at times a tumor of relatively small size pressing upon nerve structures or upon a hollow viscus such as the ureter or intestine may give rise to more pronounced symptoms than a large soft tumor extending along the long axis of the abdomen and failing to exert dislocating action upon neighboring organs or severe pressure upon nerves or vascular structures. In practically all cases the onset of symptoms is usually insidious, with indefinite abdominal pain, occasionally colicky in character, and accompanied by nausea or vomiting and a certain amount of abdominal distention. Dull lumbar pain is present in cases in which the tumor causes a dislocation of the kidney. In the event the tumor leads to interference with the free drainage of urine from the infected kidney, the patient may experience an attack of renal colic. Renal pain may also result from pressure or traction upon the renal pedicle and its sympathetic nerve supply.

Ellis[3] reported a case of a tumor surrounding the left kidney in a boy of 7, which gave rise to occasional edema of the scrotum, left lung and left side of the face.

He attributed these symptoms to probable pressure of the tumor upon the sympathetic nerve system. Osler[7] reported a case

Fig. 3. Barium enema, showing the relationship of the tumor to the transverse colon.

presenting polyuria, possibly due to pressure upon the renal vessels and solar plexus. A right sided tumor involving the perirenal structures may give rise to gastric symptoms due to pressure upon the duodenum, and also biliary symptoms through interference with the structures passing through the gastrohepatic omentum. Large tumors interfering with the vena cava or iliac veins may lead to unilateral or bilateral edema of the lower extremities. Although diarrhea had been considered an important symptom, many of the reported cases have had no interference with bowel function, and 28 per cent of the Mayo Clinic series suffered from constipation. Systemic symptoms, such as loss of weight, cachexia and anemia, depend upon the nature of the tumor, whether it be benign or malignant, and the degree of interference it offers to important intra-abdominal viscera. A common symptom is the presence of neuralgic pain in the leg due to pressure upon nerves.

The ability to palpate a retroperitoneal tumor will naturally depend upon the size of the tumor mass. Small tumors as a rule cannot be palpated. At times a good *x*-ray film will disclose the tumor but usually it is necessary to resort to indirect examination in order to establish its presence; that is, Roentgen examinations of organs secondarily involved in the growth of the tumor. In perirenal tumors a flat film of the genitourinary tract may disclose a rotation of the renal silhouette; or a distortion of the pelvis or ureter becomes evident in the course of intravenous or retrograde pyelographic studies. On the other hand, one may be able to establish the presence of such a tumor by means of a barium meal or enema, noting the presence of dislocation or compression of the various segments of the gastrointestinal tract. It has also been suggested that the use of pneumoperitoneum may be of aid in establishing a diagnosis. Cystoscopic procedures in perirenal tumors may be useful in establishing the presence of retention within the renal pelvis, or the presence of obstruction due to angulation of the ureter or ureteropelvic juncture. A film taken with the opaque catheter in situ may disclose a deviation of the ureter from its normal course from pelvis to bladder due to distortion from pressure. Abnormal elements in the urine obtained from the affected kidney simply confirm the presence of irritation of that kidney or of a superimposed infection within it. At times, the use of a lateral pyelogram may reveal a change in contour which is entirely absent in the anteroposterior film.

CASE REPORT

Mr. R. D., aged 39, married and the father of two children, was first seen April 4, 1931, complaining of pain in the right loin of one year's duration. The pain was intermittent, of a dull, aching character and was accentuated when lying on his back. There was no increase in

urinary frequency, dysuria or hematuria. The only symptom referable to the gastrointestinal tract was belching. He had lost no weight and noted no edema.

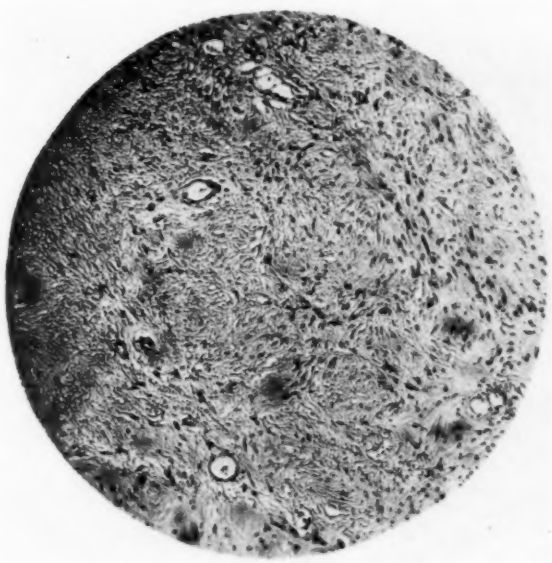


Fig. 4. Photomicrograph, low power (× 200), showing the whorl arrangement of the connective tissue cells.

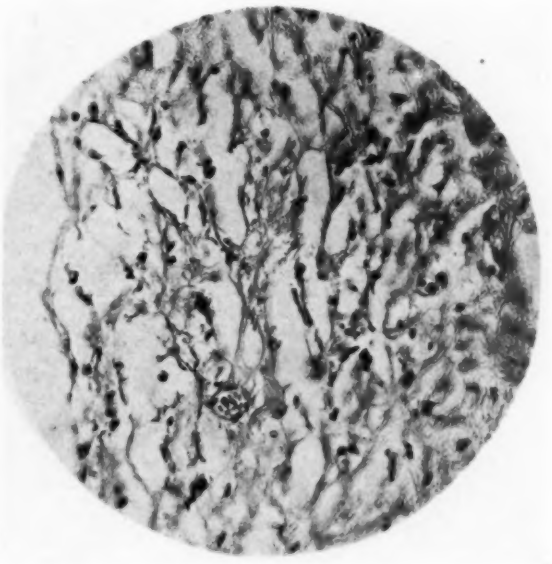


Fig. 5. Photomicrograph, high power (× 600), showing the character of the individual fibroblasts.

Five years previously he had an attack of right lumbar pain which was believed due to "gravel." The remainder of the history was entirely negative.

Save for an indefinite, small, painless, oval mass situated in the right hypochondrium, the physical examination was essentially negative.

Cystoscopy was negative. Urinalysis was negative.

X-Ray Examinations. Intravenous pyelography revealed the left kidney entirely normal. The right kidney, which was definitely diminished in size, appeared to be pushed upwards and outwards under the right leaf of the diaphragm. The pelvis disclosed a fold, giving the impression that there was something rotating the pelvis upward toward the kidney. The calices were normal, while the ureter disclosed a downward bowing, the extent of the arc being from the twelfth rib above to the third transverse process below. (Fig. 2.) There was no evidence of pelvic or ureteral ectasia.

A gastrointestinal examination failed to show any pathology in the stomach or intestines. One could clearly define an ovoid shadow about the size of a large hen's egg situated above the hepatic flexure of the colon to which it was in no way connected, and alongside the hilus of the kidney and extending from the transverse process of the first lumbar vertebra to the transverse process of the fourth lumbar vertebra. (Fig. 3.) This same shadow could be visualized on several films.

Preoperative Diagnosis. Perirenal tumor causing distortion of the pelvis of the right kidney.

Operation. On April 6, 1931, under spinal anesthesia the right kidney and perirenal space were exposed through a 7 inch Albarran incision. The kidney was slightly diminished in size and pushed upwards and outwards under the dome of the diaphragm. Situated along the mesial border of the kidney and extending from the pelvis downward below the lower pole of the kidney, there was a nonlobulated, smooth, ovoid tumor of firm consistency, about the size of a large hen's egg, fixed to the fascia of the quadratus lumborum muscle. Extending from the lower pole of the tumor there was a small duct-like structure about 1 inch in length which appeared to terminate into and blend with the fascia of the quadratus lumborum muscle. The ureter was pushed downward by this tumor. (Fig. 2.) There was no intimate connection between the tumor itself and the kidney or its pelvis and ureter. The tumor was easily dissected away from its bed and removed. The wound was closed in layers over a packing

placed into the bed of the tumor to control oozing, and a rubber tube to the renal fossa.

The wound healed by primary union and the patient was discharged from the hospital on the fourteenth day after operation.

Fig. 6. Retrograde pyelogram taken after the removal of the tumor.

Pathologic Report. Dr. H. A. Jaffe, pathologist of the Hospital for Joint Diseases reported as follows:

Gross. The specimen consists of an encapsulated retroperitoneal mass measuring $7 \times 5 \times 4$ cm. On section the neoplasm appears as a somewhat edematous and possibly mucinous yellowish-white, rather soft tumor with areas of cystic degeneration.

Microscopic. Sections show the tumor to be a fibroma with a loose, edematous, collagenous stroma as demonstrated by von Gieson stain. In places it is relatively acellular, showing only a few spindle cells. In other areas, however, there are numerous immature fibroblasts with large, plump, oval nuclei and a slightly whorled arrangement. Some of the fibroblasts are multinuclear and rich in chromatin. No mitoses are noted, however.

Many of the blood vessels contain organizing, hyaline mural thrombi and show perivascular hemorrhage. The presence of iron pigment granules within phagocytes, in their vicinity, indicates previously resorbed hemorrhage. (Figs. 4 and 5.)

Diagnosis. Retroperitoneal fibroma.

A study of the genitourinary tract following the removal of the tumor disclosed no abnormalities. The pyelogram of the right kidney was normal. (Fig. 6.) There has been no recurrence of the old symptoms up to the present writing.

SUMMARY AND CONCLUSIONS

1. About 57 per cent of retroperitoneal tumors occupy the lumbar region.

2. Retroperitoneal tumors occupying the lumbar region practically always affect the kidney, yet in spite of this, very few instances have been reported in which the symptoms were referable to the genitourinary tract.

3. When symptoms pointing to renal involvement are obtained, the tumor has caused distortion of the kidney, pelvis or ureter. Distortion may result from:

   (a) Direct pressure of the tumor upon the kidney or ureter
   (b) Retraction resulting in:
      (1) Rotation of the kidney upon its vertical or horizontal axis
      (2) Compression or angulation of the pelvis
      (3) Compression or dislocation of the ureter.

4. Retroperitoneal tumors arise most frequently from the retroperitoneal lymph nodes, connective tissue around the spine, sheath of vessels, iliac region, pelvis, and about the kidney. They may also arise from the endothelium of the lymphatic radicles around the kidney.

5. More than half of these tumors are malignant and of these, the vast majority are sarcomata.

6. Most of the reported tumors have been large.

7. Symptoms depend upon the size of the tumor and the pressure it exerts upon neighboring structures. Abdominal pain,

neuralgic pain in lower extremities, edema, constipation and the palpation of an abdominal mass are the outstanding symptoms present in the majority of cases of retroperitoneal tumors. Lumbar tumors and particularly those involving the kidney may also give rise to lumbar pain and possibly to increased urinary frequency as a result of irritation of the sympathetic nerves entering the kidney through the renal pedicle.

8. Distinctive findings in cases of perirenal tumors causing renal symptoms are: (a) renal rotation; (b) a folding type of distortion of the pyelogram; (c) dislocation or compression of the ureterogram.

9. A case is here presented of a perirenal fibroma leading to distinctive urologic findings to warrant the diagnosis of perirenal tumor prior to operation.

REFERENCES

1. Andrews, C. F. Primary retroperitoneal sarcoma. *Surg., Gynec. & Obst.*, 36: 480–485, 1923.
2. Bull. Quoted by Trout, H. H., and Meekins, G. E. Retroperitoneal sarcoma. *Surg., Gynec. & Obst.*, 31: 622–626, 1920.
3. Ellis, A. *Lancet*, Jan. 20, 1866.
4. Gobell, R. Zur Kenntnis der lateral retroperitonealer Tumoren. *Deutsch. Ztschr. f. Chir.*, 61: 1–92 (Sept.) 1901.
5. Magoun, J. A. H. *Med. Clin. North America*, Nov. 1919.
6. Mauclair, E. Tumeurs retropéritonéales prévertébrales. Clinique V, 39–41, 1910.
7. Osler, W. *Proc. Path. Soc.*, Phila., 13: 211, 1909–1910.
8. Steele, J. D. A critical summary of the literature on retroperitoneal sarcoma. *Am. J. M. Sc.*, 119: 311, 1900.

There is no evidence that urobilin is toxic. It appears to be purely an excretion product. Its reabsorption is of no known value.
From—"Bile—Its Toxicity and Relation to Disease" by O. H. Horrail (University of Chicago).

SUBPHRENIC ABSCESS WITH BRONCHIAL FISTULA

REPORT OF A CASE

FREDERICK CHRISTOPHER, B.S., M.D.

Associate Professor of Surgery, Northwestern University Medical School; Chief Surgeon, Evanston Hospital

EVANSTON, ILLINOIS

ACCORDING to Steele[1] the rupture of a subphrenic abscess into a bronchus is a relatively rare occurrence. This author was able to collect but thirty-three proved cases from the literature, although he cites Piquand[2] as having collected 112 cases in 1909: many of these, however, were not proved. Steele adds six out of a total of forty-eight cases of subphrenic abscess operated upon or autopsied at the University of Michigan Hospital from 1926 to 1936. He believes the incidence of the complication to be about 12 per cent and found its mortality to be about 50 per cent, which is about the same as the gross mortality for all cases of subphrenic abscess.

While spontaneous recovery has been reported in a few cases of rupture of a subphrenic abscess into a bronchus, immediate adequate surgical drainage is the treatment of choice (Steele).

The following case is deemed to be of sufficient interest to report:

L. R., male, age 29, was admitted to the Evanston Hospital on December 30, 1936. At 6 P.M. the preceding day, the patient was suddenly taken with very severe abdominal pain which persisted until his operation. He was seen by Dr. Frederic W. Burcky on the morning of December 30, and immediately sent to the hospital.

On admission, there was board-like rigidity of the abdomen, a leucocyte count of 18,100 and *x*-ray examination, with the patient lying on his side, disclosed a marked pneumoperitoneum. His past history was unimportant save for an appendectomy for acute appendicitis in April 1936 and the fact that for some time previous to admission he had suffered from abdominal pain coming on two to three hours after meals and relieved by soda bicarbonate.

At operation a 2 mm. perforation was found on the anterior surface of the first portion of the duodenum. A considerable quantity of turbid green fluid and pieces of food were present in the abdominal cavity. There was a fibrinous exudate upon the adjacent portions of the stomach, duodenum, gall-bladder, and liver. The perforation was closed by a linen purse-string inversion, reinforced by six interrupted linen sutures across the axis of the duodenum. Over this, omental fat was tacked in place by interrupted chromic sutures. The abdominal wall was closed as one layer with interrupted through-and-through sutures. No drainage was employed. Five hundred c.c. of citrated blood was given.

The post-operative course was at first uneventful. On the eighth day the pulse was 80 to 90 and the temperature 99 to 99.8 degrees. Three days later the pulse and temperature began to increase. A small wound infection cleared up under boric dressings but the temperature and pulse remained elevated. On the twenty-first postoperative day, coughing had become troublesome and there was pain in the right lower chest with signs of fluid. On the twenty-fourth day, there was pain in the right shoulder and the leucocyte count was 16,000. By this time, Roentgen examinations had demonstrated an unquestioned subphrenic abscess.

On the twenty-eighth day, the first stage of the operation for drainage of a subphrenic abscess was carried out. A longitudinal incision was made over the right ninth rib in the axillary line. The rib was exposed, the periosteum was shelled back and about 3 inches of rib was resected. The pleural cavity was uninvolved in the inflammatory process. The pleura on the lateral wall of the chest was sutured to that lying over the dome of the diaphragm about $2\frac{1}{2} \times 1\frac{1}{2}$ inches exposed in the floor of the wound. This area was packed with iodoform gauze.

On the next day, before the opening in the diaphragm could be carried out, the patient

began to cough up great quantities of grayish pus. The foot of the bed was immediately elevated. Despite the fact that nearly a liter of pus was coughed up, it was feared that the bronchial tree was inadequately emptied. Aspiration of the bronchi through a bronchoscope accordingly was carried out by Dr. T. C. Galloway. The diaphragm was cut through by the cautery, but the abscess cavity was not encountered. A vaseline gauze drain was placed in this subphrenic cavity, and a few days later pus began to drain from it freely. During the coughing up of pus (puroptysis) and the bronchoscopic aspiration, the patient's condition had been very poor, but thereafter he began to improve rapidly. The puroptysis subsided entirely and the thoracotomy wound healed. The patient gained some 40 pounds and regained his full health.

SUMMARY

Rupture into a bronchus is a rare but serious complication of subphrenic abscess. The possibility of this occurrence should be a factor in determining the optimum time of drainage of a subphrenic abscess. A case of subphrenic abscess with bronchial fistula is reported.

REFERENCES

1. Steele, J. D. *Ann. Surg.*, 105: 496 (April) 1937.
2. Piquand, G. *Rev. de chir.*, 39: 156, 1909.

Cavities [empyema] that cannot be obliterated spontaneously are in most instances due to improper management earlier in the course of the disease. They are usually found either in cases in which a free open drainage was instituted at too early a period or in cases in which adequate drainage had never been provided.

From—"Surgical Diseases of the Chest" by Evarts A. Graham, Jacob J. Singer and Harry C. Ballou (Lea & Febiger).

CHORIONEPITHELIOMA

J. HADLEY CALDWELL, M.D., F.A.C.S.

Senior Surgeon, St. Elizabeth Hospital, Covington, Kentucky, and Speers Memorial Hospital, Dayton

NEWPORT, KENTUCKY

CHORIONEPITHELIOMA, as the name implies, is a malignant tumor originating from the chorionic epithelium. It was first described in 1877 by Chiari, who called it carcinoma of the uterus. In 1893, Sanger called this neoplasm sarcoma uteri deciduocellulare, but it remained for Marchand in 1895 to recognize the epithelial nature as well as the placental origin of this tumor and apply the correct name.

Etiology. There is still a great deal of controversy among the leading authorities concerning the cause of these growths and their exact histologic structure. The writer will not attempt to explain all the different theories and opinions that have been advanced, except to state that it is generally conceded that this disease, in the great majority of cases, occurs sometime during the child bearing period of women following shortly after labor at term, abortion or expulsion of a hydatidiform mole; in a few very rare cases also it has been found in very young girls and elderly women. It has also been reported developing in teratoma of the testis. Most commonly, however, this type of tumor develops in the uterus of multiparous women shortly after the expulsion of a hydatidiform mole.

Pathology. *Macroscopic*. The gross appearance of uterine chorionepithelioma is fairly characteristic. The uterus in early cases is only slightly enlarged, somewhat soft and boggy, the peritoneum covering the uterus, bladder and broad ligaments is very pale and if there are no metastases present or extension of growth into the broad ligaments by continuity, the whole uterus is freely movable. In more advanced cases the tumor mass in the uterine cavity is attached to the endometrium and infiltrates deep into the muscular wall. It may be palpated when the abdomen is opened by grasping the uterus with one hand.

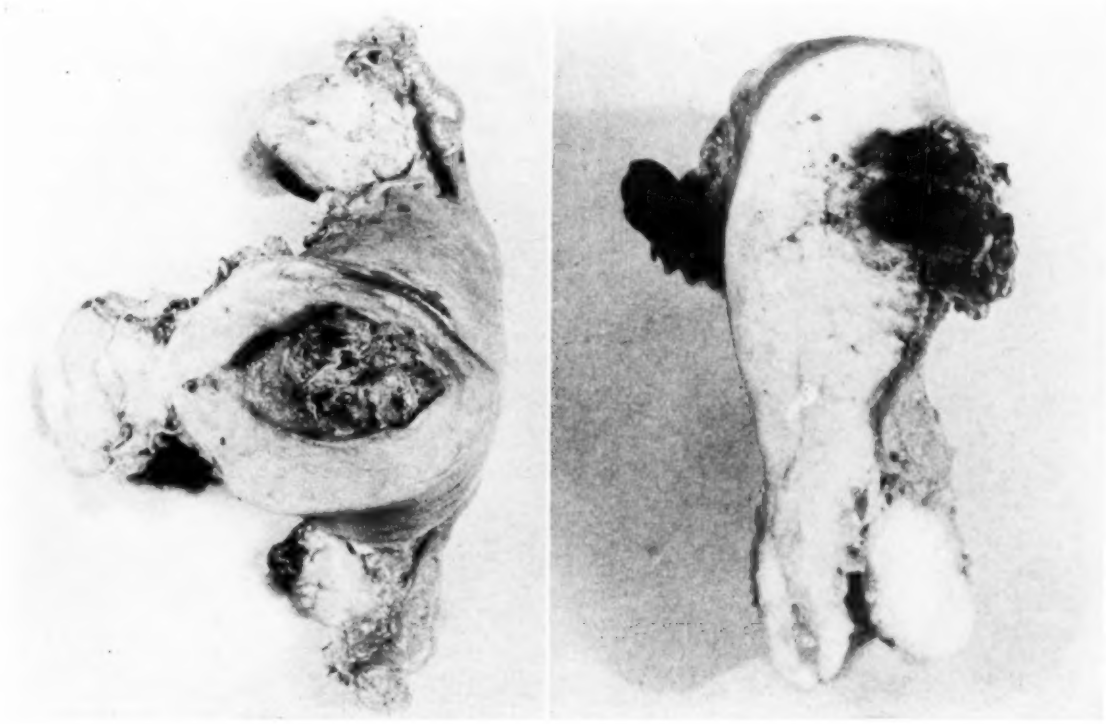
The tumors resemble either wall thrombi or interstitial nodules with black, thrombosed areas, the base of which may partly slough away. Deep-seated ulcers then develop, or more rarely, there may be a diffuse, fungating, corporeal type of growth. In spite of these variations, a distinctly hemorrhagic appearance, with infiltration of the uterine wall, is noted in all. The tumor area is friable. The growth may penetrate the uterine wall, rupturing through the peritoneal coat into the peritoneal cavity.

Ectopic. Chorionepithelioma shows a tumor having no direct anatomic connection with a previous placental site. The uterus may be perfectly normal or may show such hyperplasia of the mucosa and musculature as usually accompanies tubal pregnancy. The site of the chorionepithelioma may be in the vagina, broad ligaments or it may be intraperitoneal. The mass resembles a hematoma or collection of thrombi, tumor cells usually being found only in the periphery, with the main mass consisting of coagulum. The ectopic chorionepithelioma probably arises from a primary uterine tumor that has regressed or been expelled with a placenta or mole, or from the transplanted cells of a possibly normal intra-uterine placenta.

Chorionepithelioma of the Fallopian tube is of very infrequent occurrence. The appearance and symptoms of these cases are similar to those of tubal pregnancy, but recurrence after operation is the rule.

Microscopic. Marchand first conclusively showed that the growth originated from the tropoblasts, and that Langhans

cells and syncytium, encountered in the normal villus, also appeared in the malignant tumors. The transition from the normal villus to hydatid mole is gradual, and a further step towards malignancy is shown in chorionepithelioma. The invasive character of the growth is always present.



A B

Fig. 1. A, Case 1. Gross specimen, showing tubes, ovaries and uterus, with body of uterus opened anteriorly, containing chorionepithelioma. B, sagittal section of uterus, with chorionepithelioma shown infiltrating deep into the musculature of uterus.

Marchand has divided chorionepithelioma into two types, the typical and the atypical. The typical chorionepithelioma resembles the trophoblasts of early pregnancy. The clearly defined polygonal Langhans cells, with well-marked cell outline and large nucleus, appear mixed in with multinuclear groups of dark wandering cells of syncytium. The growth contains numerous blood sinuses, some of which harbor tumor cells as well as hemorrhagic and necrotic areas. No multiplication of villi takes place.

In the typical chorionepithelioma there is a marked tendency for the cell masses to arrange themselves in alveolar groups, to some degree resembling proliferation of carcinoma. Outgrowths of these cells invade the musculature for a considerable distance. Surrounding the hemorrhagic tumor, there is an area of fibrin and hemorrhage, and still farther toward the periphery there is a marked reaction zone.

Characteristic of this type of growth is the fact that no new formation of connective tissue or of blood vessels takes place. The blood vessels of the host are frequently penetrated and intravascular extension is not uncommon. Even a so-called typical chorionepithelioma may vary greatly in the number of Langhans cells and syncytium, the one or the other form predominating in a given portion of the same tumor or in different tumors. The Langhans cells vary more in size, shape and staining reaction of the nucleus than do normal Langhans cells. Mitosis may be present.

The atypical form generally shows a more diffuse invasion of maternal struc-

tures by large mono- or polynuclear, deeply staining cells, which may suggest sarcoma. The cells may be discrete or form

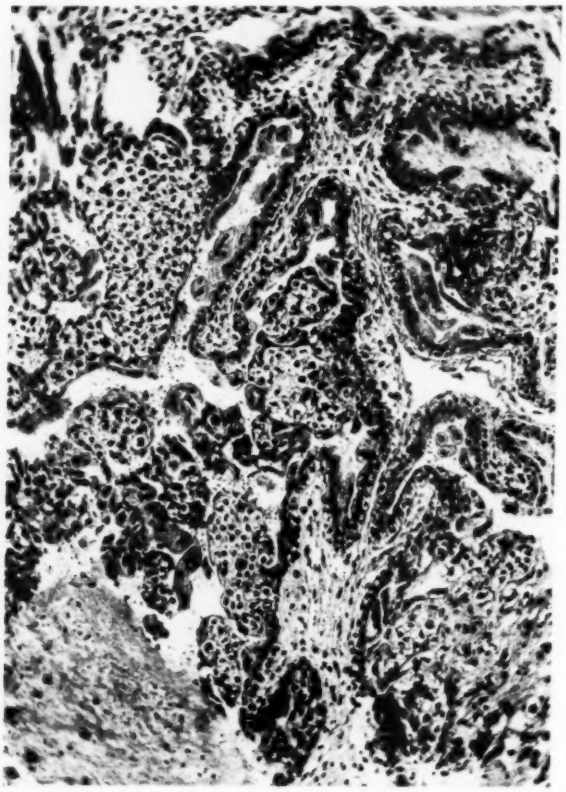


Fig. 2. Microscopic section of specimen in Case 1, showing the typical polygonal Langhans' cells.

syncytial groups and masses. Langhans cells are much less numerous than in the typical variety and may be so few as to escape observation, unless diligent search is made. As in the typical form, coagulation necrosis, fibrin, and a surrounding reaction zone are found.

From a microscopic pathologic view there are a number of subdivisions and gradations between the typical and the atypical types of this neoplasm; hence the controversy among the pathologists with reference to the diagnosis and prognosis in a given case. After an exhaustive study, Ewing has attempted to base prognosis upon histology, and in the opinion of the writer this seems logical. Ewing believes that the clinical course can be correlated with the histologic structure in such a fashion as to enable the pathologist to give a definite prognosis. This seems to be asking too much of the pathologist, but probably a relative prognosis may be given. Ewing takes a stand rather different from that of other pathologists, such as Marchand, Schlagenhaufer, Aschoff, Frank, Hirschmann and Cristoffette, who maintain that the histologic differentiation cannot be utilized in making a prognosis.

In a review of the literature, Wimpfheimer found eighty-five cases of chorionepithelioma that were classified into four types; a typical chorionepithelioma with an approximately equal number of Langhans and syncytial cells, and forty-nine cases classified by histology and treatment as follows:

		No. Cases	Well	Died	Total
Typical chorionepithelioma	Unoperated	18	1	17	
	Operated	31	18	13	49
With villi	Unoperated	5	2	3	
	Operated	5	4	0	10
Atypical Langhans cells predominating	Unoperated	2	0	2	
	Operated	4	3	1	6
Atypical syncytium cells predominating	Unoperated	12	2	10	
	Operated	8	3	5	20

From these rather scant data, we conclude that the consensus of opinion that the histologic criteria cannot be relied upon, is fully justified, except that where villi are present radical operation offers excellent hope of cure, and that syncytial tumors are fully as malignant as the typical varieties. Out of the eighty-five cases, fifty-one, or 60 per cent, died and thirty-four, or 40 per cent, recovered. The writer believes that this or any other disease that has a 60 per cent mortality is deserving of more thorough study and careful treatment.

Metastases. Metastatic extension may occur earlier in this than in any other form of tumor. Dissemination takes place through the blood stream. Metastases

have been found in almost every organ of the body, but the lungs and the vagina are the two sites of election.

Symptoms. The most important symptom of chorionepithelioma is hemorrhage. The bleeding may first appear in the early months of pregnancy, shortly after the termination of pregnancy, whether this be normal or hydatidiform, or after a period of latency existing for months or sometimes years. The bleeding is generally slight in the beginning and often repeated until profuse uterine hemorrhages take place; in fact, hemorrhage may be a terminal symptom. It sometimes simulates ruptured ectopic pregnancy, i.e., when the uterus ruptures and there is profuse intraperitoneal hemorrhage. The uterus may be found enlarged, boggy and resembling subinvolution. Subperitoneal nodules may be felt, and enlarged cystic ovaries may likewise be noted. There are generally symptoms of anemia, slight or moderate elevation of temperature, and a feeling of intense lassitude. A brief summary of a typical case would be: repeated or constant uterine hemorrhage, anemia with a low degree of sepsis, enlargement of uterus, metastases, cachexia and death.

Differential Diagnosis. Chorionepithelioma must be differentiated from ordinary non-malignant retained products of conception. Here the specimens removed with a sharp curette or a sponge forceps are rather characteristic. Such tumors as carcinoma, sarcoma and necrotic submucous fibroids may have to be excluded by microscopic section.

The most characteristic symptom of chorionepithelioma is the repeated findings of small particles of chorionic tissue in serosanguinous fluid flowing from a uterus that has been thoroughly emptied. If this symptom occurs shortly after expulsion of an hydatidiform mole, it is very suggestive of chorionepithelioma. Major importance must now be accorded to the pregnancy urine test. The writer has used the Friedman modification of the Aschheim-Zondek test with much satisfaction. In a case where the above symptoms are very definite and the Friedman test is very positive with only 10 per cent of the amount of urine usually used, one is justified in making a diagnosis of chorionepithelioma.

The incidence of chorionepithelioma, as given by different authors, varies greatly. With diligent search of the literature in 1919, Vineberg found 533 well authenticated cases. It is stated by Polloson and Violet that 45 per cent followed hydatidiform mole. The writer has personally encountered three cases, all following the expulsion of hydatidiform moles, and after review of the literature finds that few have personally treated more cases; hence the difference of opinion.

Prognosis. The virulence of this disease varies tremendously. In many instances the fulminating nature can be compared only to that of melanoma. In other cases spontaneous regression appears to have occurred, but in the opinion of the writer this seems to be a mistaken diagnosis.

Treatment. Early diagnosis is most essential. In fact, it is of the utmost importance. Where chorionepithelioma is strongly suspected, the writer would very emphatically advise against the use of the curette, since even in the hands of an expert, it is dangerous in such cases. As soon as a positive diagnosis is made, a complete abdominal panhysterectomy is indicated. In early cases, where the ovaries are normal, one ovary may be left to continue the ovarian function without danger of metastases. If a positive diagnosis of chorionepithelioma is made in young women and if there is an element of doubt, the patient should be prepared for radical operation; when the abdomen is opened, the surgeon, with the situation in perfect control, may do a hysterotomy safely, to be absolutely sure of the diagnosis. If there has been an error, the uterus may be closed and no harm has been done. If the diagnosis is confirmed by the hysterotomy, a panhysterectomy is performed at once. In all such cases in which the diagnosis is made early and the

growth is confined to the endometrium and the musculature of the uterus, the prognosis is good, if the proper operation and treatment is carried out. In inoperable cases, radiotherapy, using radium and *x*-ray, has been used with favorable results by Keene, Lackner, Wintz and others; but Hofbauer has but little, if any, faith in radiotherapy.

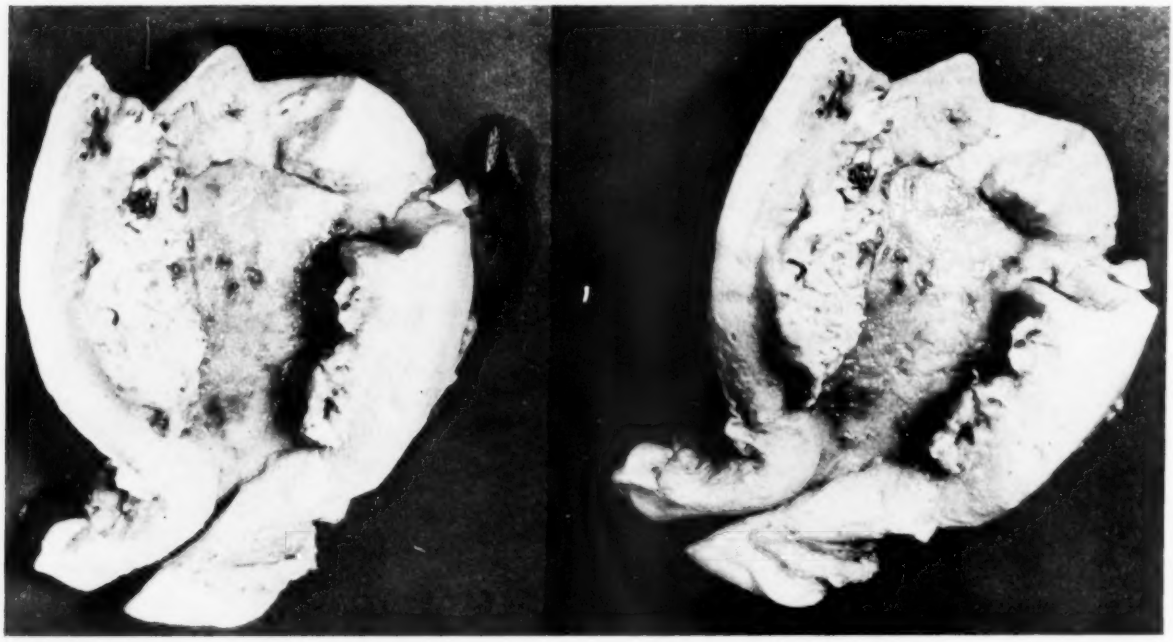


Fig. 3. Case 11. Gross specimen, showing cavity of uterus, with chorionepithelioma infiltrating deep into the musculature.

The following cases were encountered by the writer:

Case 1. Referred by Dr. J. Rolf of Covington, Kentucky, Mrs. F. N., age 39, was the mother of five children, the youngest of whom was 5. She had had no miscarriages and no previous illness. Her last regular menstruation was October 23, 1934. On December 4, 1934, she menstruated for one day. On December 18, there was profuse menstruation lasting four days, and then dribbling until January 3, 1935, Thick blood clots were passed and were followed by irregular bleeding.

I first examined her on January 9, 1935, and found the uterus in normal position, somewhat enlarged, with all the symptoms and signs of a normal pregnancy that had been threatening to miscarry. I advised rest in bed and morphine as necessary. She remained in bed a few days, then was up part of the time. She had slight uterine hemorrhage at times until February 24, when she passed a hydatidiform mole the size of a grapefruit. Dr. Rolf kept her in bed one week after this. Then she resumed some of her household duties, but soon began to bleed again when she moved about. There was a continuous serous discharge, containing occasional small pieces of chorionic tissue. This continued with occasional moderate hemorrhage until March 24, 1935. I examined her again, finding her somewhat pale and complaining of a feeling of lassitude. The pelvic organs were normal except for a slight enlargement of uterus with indication of subinvolution, as one would expect to find shortly after a miscarriage.

On March 24, 1935, a Friedman modification of the Aschheim-Zondek pregnancy test was done, using 12 and 6 c.c. of urine on successive days. The results were positive. The test was repeated three days later using only 2 and 1 c.c. of urine, and the results were again positive. The patient entered the St. Elizabeth Hospital, and three days after the second urine test, a panhysterectomy was done, removing both tubes and ovaries and covering all raw surfaces with peritoneum as usual. The appendix was also removed.

Clinical Findings. There were no adhesions. The uterus, tubes and ovaries seemed approximately normal, except that the uterus was slightly enlarged and soft. The peritoneal covering of the uterus, bladder and broad

ligaments was very pale, out of all proportion to the moderate anaemia present. A chorionepithelioma the size of a large olive was found in the fundus, attached to and infiltrating deep into the posterior wall of the uterus. (Fig. 1.)

Another Friedman test was made twelve days after operation and found negative. The patient made an uninterrupted recovery and left the hospital two weeks after operation. She has remained well with no signs of metastases up to the present writing (eighteen months after operation).

Case II. Referred by Dr. J. E. Dawson. Mrs. M. K. age 23, had been married three years and was the mother of one child, 16 months of age. Her family history was negative, and she herself had had no previous serious illness. Her menstrual history before the first pregnancy was normal, but menstruation became irregular after the birth of her baby. The last menstruation had occurred October 2, 1935; there was continuous slight bleeding from November 8 to December 20, 1935, when she passed a hydatidiform mole, accompanied by a very profuse hemorrhage.

She was removed to the hospital at that time and remained there ten days. After this hemorrhage her red cell count was 1,900,000. She improved, but continued to have a serous watery discharge with the intermittent passage of small pieces of chorionic tissue until January 16, 1936, when a moderate uterine hemorrhage occurred. Another blood count was made January 22, 1936, which revealed 3,440,000 red cells and 11,880 white. She again entered Speers Hospital on February 1, 1936. A Friedman modification of the Aschheim-Zondek test was done February 6, 1936, using only 2 and 1 c.c. of urine on successive days. The result was strongly positive. She had a slight hemorrhage February 9, 1936.

We operated February 10, 1936, doing a panhysterectomy, removing both tubes and left ovary. The right ovary, which seemed perfectly normal, was left in place, since the patient was young (23), and since the writer believes that there is no danger of metastases in this case. The appendix was also removed.

Clinical Findings. The appendix was normal; there were no adhesions; the tubes and ovaries appeared normal, the uterus slightly enlarged, soft with a patulous external os. The peritoneum covering the uterus, bladder and broad ligaments was very pale, out of all proportion to the moderate anemia present at that time. The uterus, on section, revealed a chorionepithelioma attached to and filling the fundus, infiltrating deep into the uterine wall. (Fig. 3.) Recovery was uneventful.

Fig. 4. Microscopic section of specimen in Case II, showing the presence of Langhans' cells.

The Friedman test was repeated February 26, 1936, just sixteen days after operation, using 15 and 10 c.c. of urine on successive days. This test was negative.

The patient left the hospital February 29, 1936, and has made a complete recovery. She has gained 15 pounds in weight and feels well. Another blood count was made April 16, 1936, about two months after the operation, which was as follows:

Red	5,300,000
White	9,750
Hb	70 per cent
Polys	54 per cent
Lymphocytes	35 per cent
Monocytes	3 per cent
Eosinophiles	7 per cent
Basophiles	1 per cent

Case III. This case was reported in the Cincinnati Lancet Clinic, March 29, 1914, but as it resembles the other two cases, I will review it briefly. This patient was referred to me by Dr. O. W. Brown. Mrs. M., age 20, had been married one year and had no children. She had had one miscarriage at three months in

June, 1913, and was curetted a few days later to remove pieces of retained placenta. The family history was negative. She had had no previous illness, but was neurotic and had attacks of hysteria.

Her last normal menstruation was on October 20, 1913. Three or four weeks later she began to have all the symptoms of pregnancy, with nausea and vomiting, amenorrhea, etc. She had several slight uterine hemorrhages previous to January 23, 1914, when she had a copious hemorrhage. She entered Speers Hospital, and four days later passed a hydatidiform mole. She made an uneventful recovery and returned home ten days after passing the mole. After being home two days, she began bleeding slightly again, at intervals. On February 20, she again had a profuse hemorrhage and entered the hospital. A small mass was removed from inside the fundus of uterus with a small forcep. Dr. Woolley (then Professor of Pathology, University of Cincinnati) made frozen sections of this tumor and pronounced it a chorionepithelioma and advised immediate radical operation, which was done the next day, February 22, 1914. Both tubes, ovaries, the whole uterus and the appendix were removed. The patient made a rapid recovery and is living and well at present—twenty-two years after operation. No pregnancy urine tests were made, as they were then unknown.

CONCLUSIONS

1. All patients who have expelled hydatidiform moles should be kept under observation six months or longer, with a pregnancy test done each month, this determines if any living chorionic tissue is present.

2. If there is a serosanguinous discharge from the uterus, containing particles of chorionic tissue, soon after a pregnant uterus has been emptied at or before term, it is very suggestive of chorionepithelioma.

3. According to statistics given by some writers, chorionepithelioma is very rare, and that to see three cases of chorionepithelioma, I should have to see 100,000 or 150,000 pregnancies. I do not believe that it is such a rare disease as has been pictured in the literature.

4. I believe that if general practitioners, obstetricians and gynecologists are alert, they will recognize the symptoms here described and diagnose more cases of chorionepithelioma.

5. The prognosis is good if the diagnosis is made early as it was in all three cases here reported. These patients are living and well, but the operation, as described in this report, must be performed early.

I wish to thank Dr. Alfred Glazer, pathologist at St. Elizabeth Hospital of Covington, Kentucky, for his cooperation and excellent pathological work on these cases; also to express my appreciation and thanks to Professor Hofbauer, Professor of Gynecology and Professor Richard Austin, Professor of Pathology at the Cincinnati University, for their cooperation and reading of these sections.

REFERENCES

Mathieu, Albert, and Palmer, Allen. The early diagnosis of chorionepithelioma. *Surg., Gynec. & Obst.*, 61: 336 (Sept.) 1935.

Jouravieff, A. Hydatidiform mole and chorioepithelioma. *Gynec. et obst.*, 27: 223 (March) 1933.

Curtis, A. H. Chorio-epithelioma of the uterus. *Surg., Gynec. & Obst.*, 54: 861–864 (June) 1932.

Lewis, Dean. Practice of Surgery. Vol. 11. W. F. Prior Co., Hagerstown, Md.

Davis, C. H. Gynecology and Obstetrics. W. F. Prior Co., Hagerstown, Md.

Yearbook of Obstetrics and Gynecology. Yearbook Publishers, Chicago, 1935.

Nicholson, W. P. Chorio-epithelioma. *J. M. A Georgia.* 17: 74–76 (Feb.) 1928.

Boese, A. Chorio-epithelioma. *J. Kansas M. Soc.*, 27: 302–304 (Sept.) 1927.

Sims, H. V. Choriocarcinoma (chorioepithelioma) of the uterus. *New Orleans M. & S. J.*, 83: 882–885 (June) 1931.

Hinselmann, H. Contribution to the theory of cystic mole. *Zentralbl. f. Gynäk.*, 55: 261–269, 1931.

Schaanning, C. K., Chorioepithelioma with an unusually long period of latency. *Med. Rev.*, 48: 253 (June) 1931.

Szathmary, Z. The significance of age and of roentgen and radium therapy in the treatment of mole and of chorionepithelioma. *Ztschr. f. Geburtsb. u. Gynäk.*, 98: 444, 1930.

Brews, Alan. A follow-up survey of cases of hydatidiform mole and chorion-epithelioma treated at the London Hospital since 1912. *Proc. Roy. Soc. Med.*, 28: 1213 (July) 1935.

Borras, P. E. Hydatidiform mole. *Rev. med. del Rosario*, 23: 840 (Oct.) 1933.

Stockl, E. Biologic and roentgenologic demonstration of metastasis in a case of malignant chorionepithelioma. *Monatschr. f. Geburtsb. u. Gynäk.*, 100: 33, 1935.

Fink, K. Contribution to early diagnosis of chorionepithelioma following the birth of a child capable of living. *Ztschr. f. Geburtsb. u. Gynäk.*, 83: 63–79, 1921.

SECONDARY RESECTION OF THE INTESTINE FOR RECURRENT CARCINOMA*

J. GOTTESMAN, M.D.

Adjunct Surgeon, Hospital for Joint Diseases; Associate Surgeon, Montefiore Hospital

NEW YORK CITY

MALIGNANCY of the gastrointestinal tract is a fairly common disease, comprising about 50 per cent of all malignancies. In spite of early diagnosis and early intervention, its operative removal is attended by a varying mortality of from 5 to 35 per cent in different surgeons' hands. Following successful removal, there is a high incidence of recurrence and metastatic spread. It is little wonder, then, that doctors and patients when confronted with this condition assume an attitude of defeatism, and in the presence of postoperative recurrences, a complete feeling of hopelessness.

Recurrences and metastases following resection of large intestine malignancies are unfortunately all too common, even in those portions of the large intestine that, from a technical point of view, lend themselves to easy eradication. Recurrences are prone to occur in the intestine at the site of intestinal anastomosis following resection, in the abdominal scar and in the exposed colostomy opening. This occurs especially in the exteriorization or Mikulicz type of operation. Metastatic spread occurs in the regional draining lymph nodes, over the peritoneal surface and in the thoracic and abdominal viscera.

There are few successful reports in the literature of arrests or cures of intestinal malignancy following reoperation for recurrences or metastases. Thompson recently reported several cases, and quotes Rankin and Judd of the Mayo Clinic as having had little success with attempts at secondary removal of intestinal malignancies following recurrences. The fact that such successful results can be occasionally obtained should be reported in order to encourage and stimulate surgeons to attempt reoperation in an otherwise hopelessly doomed patient. This is the purpose in presenting the following case.

CASE REPORT

Mrs. E. W. aged 55, was admitted to the Hospital for Joint Diseases on March 8, 1931. During the previous two months, she had become increasingly constipated and passed bloody and tarry stools. She also complained of loss of appetite and a feeling of weakness. There had been no pain, nausea or vomiting.

The physical examination was essentially negative. The abdomen was somewhat distended, but no masses could be felt.

X-ray examination of the gastrointestinal tract revealed a constricting annular neoplasm of the sigmoid.

On March 9, 1931, under general anesthesia, the patient was operated upon. Upon opening the abdomen, the large intestine was found distended. The circular neoplasm reported in the *x*-ray was found in the sigmoid. The Mikulicz operation was decided upon, and a loop of colon, containing the growth, was mobilized outside the abdomen, and the wound closed about it. Three days later, the afferent and efferent limbs of the intestine were sectioned with the cautery and the growth removed, leaving the patient with a double barreled colostomy.

Pathologic Report (Dr. H. L. Jaffe). Sections of the tumor showed an advanced adenocarcinoma infiltrating the muscular coat and appearing in the serosa. The tumor was fully developed and malignant. Diagnosis—papillary adenocarcinoma of the sigmoid.

The patient was discharged on March 26, 1931, and returned to the Hospital on May 5, 1931, when the spur between the limbs of the colostomy was crushed with a crushing clamp.

* From the Surgical Service of the Hospital for Joint Diseases, New York City.

The colostomy gradually closed, and the patient had normal bowel movements. After several weeks, the colostomy reopened.

On September 14, 1931, the patient reentered the Hospital for a closure of the colostomy opening. After excising the scar and separating the mucosal edges of the intestine, the stoma was closed with inverting Lembert sutures. The skin and fascia were closed. A hard nodule excised from the intestinal mucosa was reported carcinoma.

Following this operation, the fistula remained closed for several months and then again reopened. A nodule which gradually increased in size, was noticed in the colostomy opening. Most of the fecal discharge was through the colostomy opening, with occasional bowel movements. In August 1932, radium needles were inserted into the recurrent malignant nodule. There were no visible effects, the tumor continued to grow, and a further radical removal was decided upon.

On January 24, 1933, under general anesthesia, an elliptical incision was made around the fistulous opening, and this incision was extended into the peritoneal cavity. The afferent and efferent limbs of the colostomy were resected together with the elliptical portion of the abdominal wall containing the recurrent growth. An end-to-end anastomosis of the intestine was performed, the abdominal wall closed in layers, and rubber dam drains inserted to the site of the anastomosis. The wound healed by primary union, the drains being removed after eight days. The mass was reported to be adenocarcinoma of the intestine, recurrent. The patient made a good recovery, and has continued well to date, with normal functioning bowel movements.

The unusual outcome in this case as the result of rather fortuitous circumstances suggests that it might be deliberately worthwhile maintaining an open colostomy for a considerable length of time following a Mikulicz operation. Since many recurrences take place in the intestinal wall at the site of amputation of the gut, it would be very easy to detect such recurrences if the intestinal ends were kept exposed in the colostomy opening. This happened in the above case. If, after a reasonable period, no recurrences are demonstrable, it would be safe to close the stoma and reëstablish the intestinal continuity. The efferent portion of the intestine could be kept patent with irrigations and enemata.

This patient died on January 21, 1938. For a number of months prior to death she complained of pain in the back, chest and lower quadrant. Autopsy showed a generalized metastatic spread, involving the lungs, liver and retroperitoneal lymph-nodes.

SUMMARY

1. A case of secondary resection of the intestine for recurrent carcinoma, well six years after operation, is presented.

2. The suggestion is made to deliberately maintain a functioning colostomy in order to detect recurrences in the intestinal wall. If none occur after a reasonable period of observation, closure can be attempted.

REFERENCE

1. Thompson, J. W. *J. A. M. A.*, 107: 1688 (Nov. 21) 1936.

MESENTERIC CYSTS CAUSING INTESTINAL OBSTRUCTION*

J. W. GALE, M.D. AND J. L. KEELEY, M.D.

MADISON, WISCONSIN BOSTON, MASSACHUSETTS

AT first thought there seems to be very little justification for reporting a single case of an established disease entity from which no significant conclusion can be drawn and which resembles previously reported cases in most of its principal features. However, in dealing with a comparatively rare condition it is well to realize that only through the accumulation of such data will the true clinical manifestations become apparent. It is with this thought that the following case report is submitted.

Since Benvieni,[1] in 1507, first reported mesenteric cysts, the literature has been increased by reports of either single instances or small series of cases. Dowd,[2] in 1900, stated: "Reports indicate that mesenteric cysts are being removed as often as once a month." He himself reported 145 such cases. Higgins and Lloyd,[3] in 1924, numbered the reported cases at about 245. Miller[4] stated that there were less than 400 cases of all types recorded up to 1935.

The main features of interest are the theories of origin and formation, and the complications which develop. The former find expression in the many classifications, while the latter determine to a large extent the clinical picture.

Braquahaye,[5] in 1892, offered a classification which included the following types of mesenteric cysts: (a) sanguinous; (b) lymphatic cysts, including chylous; (c) hydatid; (d) congenital and dermoid; and (e) cysts of adjoining organs, such as the ovaries, pancreas, etc. His classification was followed shortly by that of Moynihan's,[6] in 1897, which listed serous, chylous, hydatid, blood and dermoid cysts, and cystic malignant disease.

Dowd divided the cysts into three principal groups:

I. Embryonic, including: (a) dermoid; (b) serous; (c) chylous, and (d) hemorrhagic, and still others with walls like those of the intestine.

II. Hydatid cysts.

III. Malignant disease.

The collection of many types of cysts under the heading embryonic had its influence in the classifications which followed. Ayer added two groups to Dowd's classification as follows: (a) cysts arising from the glandular structures in the intestinal wall; and (b) cysts of normally placed retroperitoneal organs.

Niosi[7] gave the following classification for cysts of the embryonic group: 1. cysts of intestinal origin: (a) sequestration from bowel during development; (b) from Meckel's diverticulum. 2. Dermoids. 3. Cysts from retroperitoneal organs (germinal epithelium, ovary, Wolffian body, Mullerian duct).

Gould's report,[8] in 1915, did not include cysts, but did present tumors of the mesentery, all of which had walls of dense fibrous tissue without an epithelial lining. He thought these resembled very closely the "cold abscesses of the mesentery" which were reported by Beatson (1908), Kukula (1899), and Baum (1902). Tubercle bacilli were found in the contents of the cystic structure reported by Bealson and Kukula, but were not present in any of Gould's cases. Giant cells were also absent.

Swartley[9] writes; "It is plain that more and more are coming to regard all mesenteric cysts not parasitic or malignant, as of embryonic origin. The term 'cystic malignant disease' which has been given as a class of mesenteric cysts is no longer tenable. Malignant cysts are probably for the most part, originally, simple cysts which subsequently have become malig-

* From the Department of Surgery, University of Wisconsin Medical School.

nant. Moreover, a metastatic malignant tumor of the mesentery, or for that matter a primary one which undergoes cystic degeneration is not morphologically a cyst."

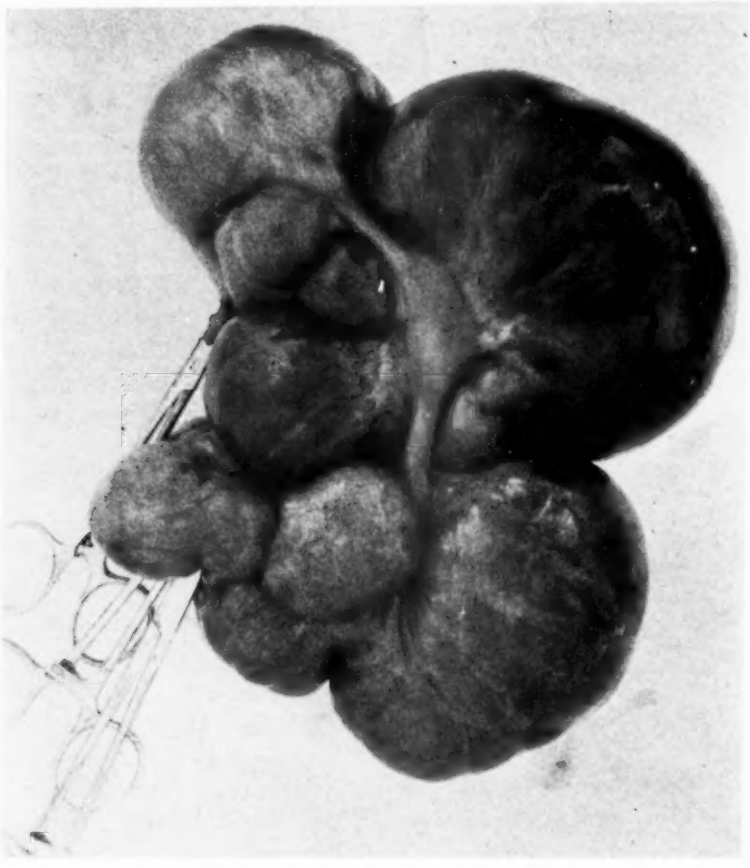


Fig. 1. Photograph of the specimen showing groups of cysts adjacent to the bowel, a portion of which is seen in the center of the illustration. A more complete description is given in the text.

Ribbert's expression of the theory that sequestration of a portion of embryonic tissue or organ may occur and develop in an anomalous manner is discussed by Miller.[10] That this course of events may occur is supported by Dowd, who pointed out the many instances of accessory structures such as lobes of thyroid and thymus glands and breast tissue. Accessory spleens and double spleens have also been reported and an accessory pancreas is occasionally found.

In 1908, Lewis and Thyng[11] described the "regular occurrence of intestinal diverticulae in the embryos of the pig, rabbit, and man." Hueper,[12] in 1927, wrote of finding a cyst in the subserosa of the wall of the duodenum of one of thirty rabbits and cats examined with this anomaly in mind. The cyst had a well developed musculature and mucosa similar to that of the duodenum, but the layers were thinner.

There seems to be little doubt at present that the mechanism of sequestration of intestinal diverticulae is responsible for many mesenteric cysts. Such a process would lead to formation of cysts, the walls of which resemble very closely those of the adjacent bowel. By a shift in position from the anti-mesenteric border of the gut, Meckel's diverticulum and remnants of the omphalo-mesenteric duct may come to lie between the folds of the mesentery and give rise to cysts in the walls of which

may be found layers similar to those of the gastrointestinal tube.

Dermoid cysts which are intra-abdominal are said to originate in the ovary. Swartley, in 1927, stated that dermoid cysts of the mesentery have been reported thirty-five times. Since all of these have been found in women some authors conclude that their origin is always ovarian.

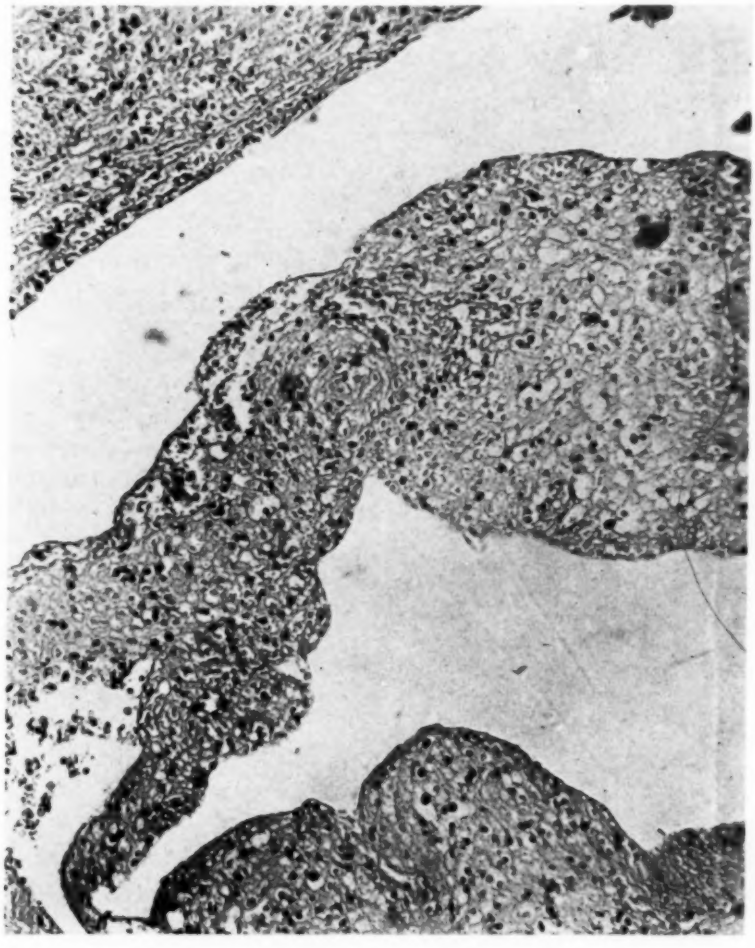


Fig. 2. Photomicrograph of cyst wall showing hyaline and amorphous material. There is a definite limiting membrane lining the cyst.

The general picture becomes somewhat clarified when such a classification as that of Higgins and Lloyd is preceded by their definition of true mesenteric cysts, i.e., "those which occur in or near the mesentery and which are not malignant, dermoid, or parasitic and do not arise in any normally placed retroperitoneal organ." Thus cysts of embryonic origin arising from mesodermal remnants incarcerated behind the developing peritoneum and subsequently coming to lie between the peritoneal layers of the mesentery constitute one class of true cysts. The others are those of intestinal origin and include cysts arising in most cases from diverticulae of the bowel during the development of the gut, and those which may develop from the persistent portions of the vitelline duct. Those of the latter group are usually identified by microscopic examination. The wall has layers of smooth muscle arranged similarly to those of the intestinal tract. The epithelial lining is dependent somewhat on the degree of tension in the cyst. Simple columnar epithelium is often seen, occasionally stratified layers are present, and cases have been reported in which ciliated epithelium lined a mesenteric cyst.

The presence of goblet cells in the lining of a cyst of this group has been interpreted as additional evidence of intestinal origin.

The other group includes those which have been called serous, lymphatic, chylous or hemorrhagic, according to their contents. Dowd suggested that the contents of such cysts should not be used as a means of classifying them. Both lymph spaces and blood vessels have been identified in the walls of mesenteric cysts. Hemorrhage accounts for the contents of the hemorrhagic cysts. Chyle effuses in chylous ascites and its passage into a cyst cavity from the wall seems to be an example of the same process. In regard to the serous cysts Dowd states, "Whether the fluid is exuded from the blood vessels in the form of serum, or whether it is secreted from the cells which line the cyst seems to be largely dependent on the structure of the cyst wall." Microscopically the wall of cysts of this group is composed principally of fibrous tissue in which round cells may be found. The lining is usually a single row of epithelial cells, but destruction of the lining by inflammation may occur. The small bowel is the site of more than half the reported cases. The mesentery of the ileum contains about one-fourth of the total number.[17] Single cysts are more numerous, the number of multiple cysts being comparatively small. As in other abdominal cysts extremes of size have been observed. The unilocular and multilocular types appear to be equally numerous.

CASE REPORT

A white male, aged 14 years, was admitted to the Wisconsin General Hospital July 7, 1934, with a chief complaint of abdominal pain. He had experienced this symptom at intervals since the age of 9. The pain was sharp and cramp-like and occurred usually about an hour after meals. The duration of the attacks was variable, some of them subsiding after a few days while others persisted for weeks. On the advice of his physician he had altered his diet, taking only liquids and soft food in smaller quantities than usual, but with more frequent feedings. This régime afforded moderate relief. The patient had vomited with the first attack, and also during the attack for which he was hospitalized. During the intervening period of five years the seizures of abdominal pain had not been accompanied by emesis. The attacks started after scarlet fever in 1927. In 1930, Roentgen ray pictures of the stomach were taken and an ulcer was thought to be present. Many types of ulcer treatment were instituted, none of which altered his abdominal condition. There had been a weight loss of 20 pounds since April 1934. The remainder of the history was not significant except that mild constipation had persisted over a period of years for which mineral oil was given.

The pertinent findings on physical examination were confined to the abdomen. There appeared to be no distention, but generalized increased tone of the abdominal muscles, especially the recti, was noted. There was slight tenderness in the entire epigastrium. No masses were palpable. The liver edge reached the costal margin. There was no fever. The pulse and respiratory rates were within normal limits.

The urinalysis on admission revealed a specific gravity of 1.029. The urine was acid in reaction; showed a faint trace of albumin and no glucose, but was positive for acetone. Microscopic examination showed three fine granular casts per ten low power fields; occasional squamous epithelial cells, a few bacteria, and one to two white blood cells per high power field. The blood count was as follows: hemoglobin 90 per cent; red blood cells, 6,160,000; white blood cells, 13,150; neutrophiles, 70 per cent; eosinophiles, 2 per cent; and lymphocytes, 28 per cent. The Wasserman reaction was negative. The blood sugar was 77 mg. per cent, and the non-protein nitrogen was 30 mg. per cent. A tentative diagnosis of pyloric obstruction was made.

During the period of observation the urine was examined for lead on two separate occasions and none was found. Through the fluoroscope, barium, which had been administered to the patient elsewhere prior to his admission, was visualized in the upper loops of small bowel, and a diagnosis of high intestinal obstruction was made. The cause of the intestinal obstruction was undetermined prior to operation.

On July 14, 1934, under cyclopropane anesthesia an exploratory laparotomy was per-

formed. The abdomen was opened through a mid-right rectus incision and marked dilatation of almost the entire jejunum was seen. A mass was brought up into the wound from the lower part of the abdomen. This mass consisted of several rather large dilatations which were at first thought to be diverticuli of the small bowel. Considerable constriction of the bowel lumen was noted, and because of this finding a resection of approximately 45 cm. of jejunum was done. An end-to-end anastomosis was made and a catheter was inserted into. the dilated jejunum above the anastomosis. About 500 c.c. of intestinal contents were removed from the dilated bowel, rendering closure of the abdomen much less difficult. The catheter was brought out through the lower end of the incision which was closed in layers without drainage. The postoperative course was essentially uneventful except for moderate abdominal pain and the development of a mild cystitis. On July 27, eleven days after the operation, the catheter was removed and the jejunal fistula healed rapidly. The patient was discharged from the hospital on August 4, 1934.

The description of the specimen (Fig. 1) removed at operation was as follows: "Specimen consists of a piece of small bowel about 45 cm. in length attached to which are several cysts, the largest being $11 \times 8 \times 6$ cm., another $9 \times 8 \times 5.5$ cm. also being present. These cysts can be divided into four groups, each consisting of one large and several smaller cysts. There is communication within the groups, but not between groups. There are several small cysts 1 to 2 cm. in diameter which are separate. The cysts are in the mesentery and do not communicate with the bowel. They are surrounded by a thin fibrous wall and contain brownish fluid without noticeable odor. The cysts are unilocular. Within the bowel wall there is a small firm tumor, 1×1.5 cm. Microscopically the section shows numerous spaces varying greatly in size and lined by a single row of cells."

SYMPTOMATOLOGY AND DIAGNOSIS

It may be said that the symptoms of mesenteric cysts depend on conditions which most authors list under the heading of complications. The most characteristic symptom complex includes repeated attacks of abdominal pain, usually cramp-like and accompanied by nausea, vomiting and often abdominal distention. Constipation may be present, and may alternate with or be followed by diarrhea. This sort of history is given in those cases where the encroachment on the bowel lumen has occurred by pressure of the cysts from the outside. Volvulus, intussusception, adhesions or kinking may cause complete obstruction, necessitating operation, at which time the cause of the obstruction may be determined.

The physical examination may aid in the diagnosis if a smooth, rounded, cystic and freely movable mass is palpable. Similar findings are present in cases of omental cysts, but Porter's[13] observation that pain and nausea or vomiting are associated with mesenteric cysts more often than with any other intra-abdominal cyst should help in differentiating omental from mesenteric cysts.

Women are more often affected than men. Most cases are seen in the fourth decade, although Palmer's[14] case and one reported by Sala and Nachmie had prenatal volvulus from a mesenteric cyst. Volvulus caused by mesenteric cysts was present in Miller's case (four days old), and in Cutler and Merriam's case[15] (three and one-half months). Old age is no assurance that an intra-abdominal lesion is not a mesenteric cyst. Many have been discovered in the fifth and sixth decades.

The lack of general wasting has been considered to be an important point in diagnosis. Jones'[16] patient, and one in the accompanying case report each lost about 20 pounds in the few months prior to operation. These instances, however, represent the exception rather than the rule.

If inflammation occurs in the cyst the differential diagnosis must then include the usual acute abdominal conditions which are accompanied by signs of peritoneal irritation and constitutional reaction.

Swartley lists the complications as follows:

1. Obstruction.
2. Peritonitis as a sequel to obstruction.

3. Hemorrhage into the cyst. This has been known to be fatal.
4. Rupture of the cyst.
5. Torsion of the cyst.
6. Pressure upon organs if the cyst becomes impacted in the pelvis.

Miller writes of the group of cases which do not have any of the above complications and states that the signs and symptoms in this group are not so uniform. Among them are abdominal fulness, pressure, dragging sensations in the abdomen and, occasionally, history of recurrent attacks of very mild intestinal obstruction.

Most authors state that the correct diagnosis has never been made prior to operation, and that this is an indication of the diagnostic difficulties encountered. Warfield,[17] who reviewed the literature from 1920 to 1932, calls attention to correct diagnosis reported by Haworth (1920), Bertolini (1921), Naumann (1921), Levinson and Wolfsohn (1926), Ciarlo (1927), Aloi (1927) and Finucci (1930). Parker,[18] in 1923, made the correct diagnosis from Roentgen ray and fluoroscopic examination. The preoperative diagnosis in the case reported by Hueper was acute appendicitis and mesenteric cyst. The appendix was not found to be acutely inflamed at operation, but the cyst was present in the mesentery of the ileum.

TREATMENT

Treatment has received but scant attention in the literature for the reason that it does not differ in the acute cases from that of intestinal obstruction or peritonitis from any other cause.

Enucleation of the cyst or cysts is the operation of choice if it can be done without damaging the blood vessels of the mesentery. Resection of the involved portion of bowel may be necessary. It is accompanied by a high mortality (60 per cent), but such figures include acute cases in which the mortality would be high for bowel resection done for obstruction from any other cause. The acute cases are very ill of a high intestinal obstruction when the operation is decided upon, so that the high mortality rate is not surprising. Drainage of the cyst may be of value temporarily, but the danger of sinus formation which may ultimately require excision should be remembered. Aspiration and marsupialization are both obsolete, but might be useful in certain instances.

SUMMARY

1. A case of mesenteric cysts in a male aged 14 years is reported. The history in this instance was one of recurring attacks of intestinal obstruction over a period of five years, the final attack necessitating surgical intervention. The cause of the obstruction was not determined prior to operation.

2. Resection of the involved bowel was done and an enterostomy above the site of the end-to-end anastomosis was used to decompress the dilated loops of intestine. The enterostomy tract closed promptly and the patient recovered.

3. The case is believed to be one of true mesenteric cysts arising from mesodermal remnants.

4. Two additional instances found in the literature of correct diagnosis of mesenteric cysts before operation or autopsy are pointed out.

REFERENCES

1. Benivienius, A. De abditis nonnullis ac mirandis morborum et sanationum causis. Florence, Italy, 1507. In: Galen, D. Liber de plenitude. 4°, Basileae, 1528, pp. 1–21.
2. Dowd, C. N. Mesenteric cysts. *Ann. Surg.*, 32: 515–542, 1900.
3. Higgins, T. T., and Lloyd, E. I. Mesenteric cysts. *Brit. J. Surg.*, 12: 95–105, 1924.
4. Miller, L. A. Rarity of congenital cysts. *Am. J. Dis. Child.*, 50: 1196–1202, 1935.
5. Braquahaye, J. Des kystes du mésentère. *Arch. gén. de méd.*, 2: 291, 1892.
6. Moynihan, B. G. A. Mesenteric cysts. *Ann. Surg.*, 26: 1–30, 1897.
7. Niosi, F. Die Mesenlerialcysten embryonalen Ursprungs nebst einigen Bemerkungen zur Entwicklungsgeschichte der Nebennieren-Rindensubstanz sowie zur Frage des Chorionepithelioms. *Virchow's Arch. f. path. Anat.*, 190: 217–338, 1907.
8. Gould, E. P. Three mesenteric tumours. *Brit. J. Surg.*, 3: 42–47, 1915.
9. Swartley, W. B. Mesenteric cysts. *Ann. Surg.*, 85: 886–896, 1927.

10. Miller, R. T., Jr. Enterogenous mesenteric cysts. *Johns Hopkins Hosp. Bull.*, 24: 316–322, 1913.
11. Lewis, F. T., and Thyng, F. W. The regular occurrence of intestinal diverticula in embryos of the pig, rabbit, and man. *Am. J. Anat.*, 7: 505–519, 1908.
12. Hueper, W. Mesenteric enterocystoma. *J. Lab. & Clin. Med.*, 12: 427–429, 1929.
13. Porter, M. F. Chylous cysts of the mesentery. *Ann. Surg.*, 43: 380–389, 1906.
14. Palmer, D. W. Mesenteric cyst with prenatal volvulus. *J. Med.*, 8: 239–241, July 1927.
15. Cutler, G. D., and Merriam, J. C. Intestinal obstruction in infant due to mesenteric cyst with volvulus. *Boston M. & S. J.*, 193: 550–552, 1925.
16. Jones, E. G. Cysts of the mesentery. *Surg. Gynec. & Obst.*, 21: 56–60, 1915.
17. Warfield, J. O. Jr. A study of mesenteric cysts. *Ann. Surg.*, 96: 329–339, 1932.
18. Parker, C. H. Report of three unusual abdominal cases. *Am. J. Roentgenol.*, 10: 605–610, 1923.

Bile, under ordinary conditions, is sterile, but is not bactericidal. . . . Jaundice is a symptom, not a disease.
From—"Bile—Its Toxicity and Relation to Disease" by O. H. Horrall (University of Chicago).

NON-RECURRENT DISLOCATION OF THE PATELLA

IRVIN BALENSWEIG, M.D.

Assistant Professor of Orthopedic Surgery, Cornell University Medical College

NEW YORK CITY

THE following case report is that of a chronic dislocation of the patella, which existed for twenty-three years before operation. Recurrent or habitual dislocation of the patella may result from a congenital abnormality and invariably the patella dislocates laterally. It is usually accompanied by a poor development of the lateral femoral condyle and is most frequently observed in females. The patient soon learns to reduce the patella without assistance. The patella may also be dislocated as result of traumatism when it is usually dislocated medially or rotates on its long axis and becomes engaged between the condyles of the femur.

The following case report is that of a chronic dislocation of the patella of the non-recurrent type:

FIG. 1. Anteroposterior view of knee joint, revealing lateral dislocation of patella.

FIG. 2. Lateral view of knee joint prior to operation, showing absence of patella from its normal location.

G. M., aged 49, was first observed on November 17, 1929, when she complained of weakness of the left knee. She stated that she was quite well until at the age of nineteen years she was suddenly taken ill with what was then diagnosed as acute articular rheumatism. The left knee had become quite swollen and after several months she was allowed up and about

with the aid of crutches. Since that time, she states, the knee frequently "gave away." Two weeks prior to the initial observation she fell

fixed laterally. There was no evidence of periarticular thickening nor any evidence of arthritis in any of the other joints of the body.

Fig. 3. Anteroposterior view following operation.

Fig. 4. Lateral view following operation.

when her knee gave away and she had had pain and disability since. Several years ago she had sought surgical attention, but was then informed that nothing could be done.

Examination. The patient was observed to be in excellent general condition. She walked with a left knee limp and maintained the left knee in an attitude of partial flexion. She walked with the aid of a cane and with an elastic bandage about the knee.

Clinically, it was observed that the left knee was wider than its fellow and flattened anteriorly, and that the patella was not situated in its normal location. The patient could voluntarily extend the knee joint to 155 degrees; passively it could be extended to 180 degrees. The angle of greatest flexion was 75 degrees, whereas the right knee could be flexed to 45 degrees. There was approximately 15 degrees of lateral instability of the joint and marked atrophy of the quadriceps group. The patella was found to be dislocated and

The *x*-rays disclosed the presence of a lateral dislocation of the patella with a partial subluxation of the tibia and osteophytic production about the condyles of both the tibia and femur. The joint space was narrowed. The tibial condyles were flattened and the spinous processes were very much shorter than normal. In the lateral view the patella could not be observed. Otherwise the joint appeared to be well formed.

The patient was admitted to the Hospital for the Ruptured and Crippled and on December 2, 1929, operation was performed. The joint was prepared in the usual manner and a curved incision made about the knee with the convexity downward, exposing the joint in its entirety as well as the tibial tubercle. The patella was identified and found to be displaced outwardly and firmly adherent in this position. The ilio-tibial band was contracted and tense. This latter structure was then sectioned transversely 1 inch above its distal attach-

ment as were all the contracted tissues on the outer side of the knee joint. It was only after such sectioning that the patella could be replaced in its normal location. The tibial tubercle was then freed from its attachment into the tibia and implanted well towards the medial side of the tibia into a channel of bone previously prepared. An incision was then made on the inner side of the capsule of the knee joint and the capsular structures overlapped and sutured, thus plicating the redundant tissues. Following this procedure a rather large opening was left on the outer side of the joint. This was repaired by removing a section of fascia lata from the same thigh and suturing it into the defective area. The wound was closed throughout, using fine chromic catgut sutures, and a plaster of Paris bandage was applied from the toes to the groin, maintaining the knee in approximately 15 degrees of flexion.

Postoperative *x*-rays revealed the presence of a normally situated patella.

The patient made an uneventful recovery and was discharged from the hospital on December 21, getting about with the aid of crutches. She was subsequently observed in the office, where she received physiotherapy, graduated exercises, and was finally discharged from active treatment on February 25, 1930.

Fig. 5. Anteroposterior view with patient standing.

Fig. 6. Lateral view, revealing power of voluntary extension of the knee.

Some six months after operation she exhibited a range of motion of from 170 degrees of voluntary extension to 50 degrees of voluntary flexion. The quadriceps muscle, though weak, was sufficiently strong to maintain balance. She was well able to walk up and down stairs without external assistance.

When last observed on June 23, 1936, she stated that she had progressed very favorably and only on rare occasions was it necessary to rebandage the knee to give her some additional support. Examination at this time

revealed a well functioning knee joint with a range of motion from 175 degrees of extension to 50 degrees of flexion. The patella was freely movable and was situated in its normal location, and the knee was perfectly free from any arthritic manifestations.

It is well recognized that the patella is not essential for normal life when the quadricep tendon is attached through the patella tendon into the tibial tubercle. There are occasional instances recorded in the literature of absence of patellae; the writer has had occasion to examine and treat a child of five years who had no evidence of either patella on clinical or Roentgen ray examination, and whose father had extremely small patellae with very large tibial tubercles. Such individuals get about quite well insofar as the strain of the quadriceps pull is transmitted directly to the tibial tubercle, and not through the patella. The leverage of the extensor apparatus is greatly enhanced by the interposition of the patella. Contraction of the quadriceps group tightens not only the tendinous insertion and pulls the patella forward, but it also puts tension on the anterior reinforcing apparatus of the knee, and because of this fact becomes a most important stabilizer of the knee in extension. From this point of view the patella serves a very useful function for without it the dynamics of the knee joint is altered and the individual so afflicted has difficulty in performing arduous tasks; in running; in walking up and down stairs. On level surfaces the function is but little interfered with.

The above operation has distinctly improved the dynamics of the knee joint and the patient has now been observed for a period of seven years during which time the patella has remained in its normal position.

This case is recorded as it not only describes a surgical rarity but also presents a reconstructive operation which has withstood a test of several years.

PELLEGRINI-STIEDA'S DISEASE

A CASE REPORT

MEYER O. CANTOR, M.D. AND ELDEN C. BAUMGARTEN, M.D.

DETROIT, MICHIGAN

FOLLOWING a single or multiple traumatization of connective tissue, muscle or a ligamentous structure, when an area of calcification appears, the condition is known as myositis ossificans. If, however, the tibial collateral ligament is the injured part and the characteristic semilunar calcification appears in it, usually along the superior surface of the medial condyle of the femur, then the condition ceases to be myositis ossificans and becomes Pellegrini-Stieda's disease.

We feel that there is no difference between this and the calcification that occurs in the shoulder muscles of soldiers from the constant trauma of the rifle, "infantry-man's shoulder," the calcifications that occur in the leg muscles of the cavalry-man from the trauma of the saddle against the adductor muscles, "cavalry-man's myositis," and the calcified areas which are seen so often in the gastrocnemius muscles of ballet dancers from the microscopic injuries sustained in these muscles as a result of the constant strain imposed upon them by this dance. Innumerable similar localized areas of calcification can easily be called to mind.

Our case report lends further corroboration to the conception of the American writers, Kulowski,[1] Riebel and Riebel,[2] Oxford,[3] and Wetzler and Elconin,[4] that the process is a metaplasia in this specific connective tissue structure to form bone as a result of trauma. That all connective tissue may respond to trauma in this way is a well recognized fact. The great importance of this specific lesion lies in the fact that the majority of such injuries are sustained at work and become therefore compensation cases. The early recognition of the lesion is most essential for successful treatment. That a great many such cases are undiagnosed is unquestioned. The general practice is to *x*-ray the part immediately following the injury. Since in these cases such *x*-rays show no pathology, the patients complaints two months later, when the calcification occurs, are often attributed to malingering. An *x*-ray taken at this time, however, shows the characteristic lesion. A failure to take such check-up *x*-rays might result in a considerable monetary loss to the employer.

Very few cases of this interesting finding are reported in the American literature, although the European literature has many such case reports. This is apt to give one the erroneous impression that the disease, if it can be called such, is rare. Consultation with several of the outstanding industrial surgeons in Detroit disclosed on the other hand, that they regarded this calcification in the tibial collateral ligament described by Pellegrini and Stieda as something so commonly seen following trauma to the knee joint that no importance was attached to it. It is considered the same as myositis ossificans would be elsewhere, and not as a disease entity per se.

In all these reported cases three to four months and sometimes longer elapsed from the time of the injury to the time that the diagnosis was made. In our case report only eight weeks elapsed from the time of the injury until the typical *x*-ray findings pathognomonic of this disease were found. This is one of the earliest on record. From our *x*-ray findings it is very likely that the lesion developed two to three weeks previously. This would fix the time of development of the calcification to five weeks following the trauma, at which time it would have been possible to diagnose it by *x*-ray.

CASE REPORT

G. T., a colored male, aged 33, was struck a glancing blow upon the left shoulder by the

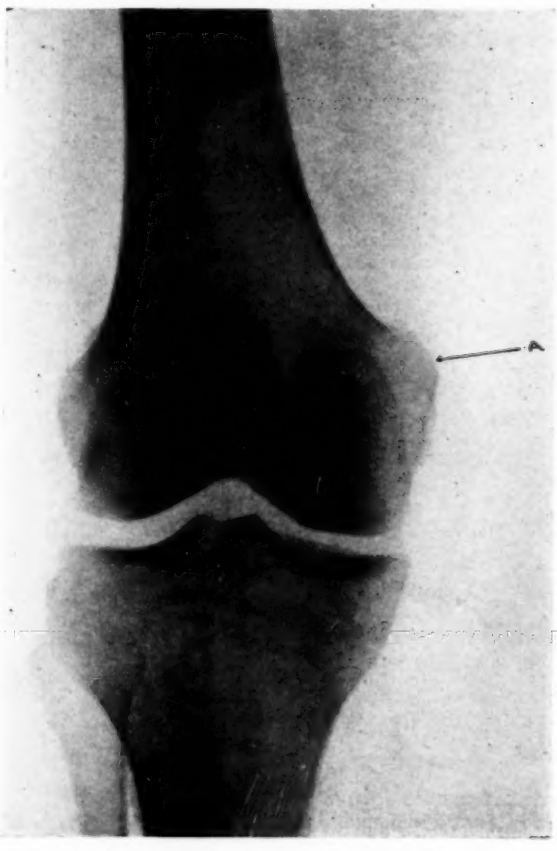


Fig. 1. *X*-ray taken four days after injury. Note point A. No injury to medial condyle of femur.

collapse of a plaster wall. He was thrown to the ground, but arose and went about his business, although he complained of some soreness in the left knee joint. He continued being ambulatory for the next four days, but then he called one of us (M. O. C.) because of swelling and pain in the left knee joint which had become progressively worse.

Upon examination at this time (October 5, 1936), the left knee was found to be considerably swollen with a definite area of tenderness over the tibial collateral ligament. There was no hypermobility of the joint. Weight bearing was painless if the leg was not moved as to produce lateral motion at the knee. The maximum point of tenderness was found to be over the superior surface of the medial condyle of the femur. The patella was floating. The findings characteristic of fluid in the knee joint were easily elicited.

The patient was hospitalized and *x*-rays were taken of the knee joint on the same day. The Roentgen findings disclosed no bony pathology.

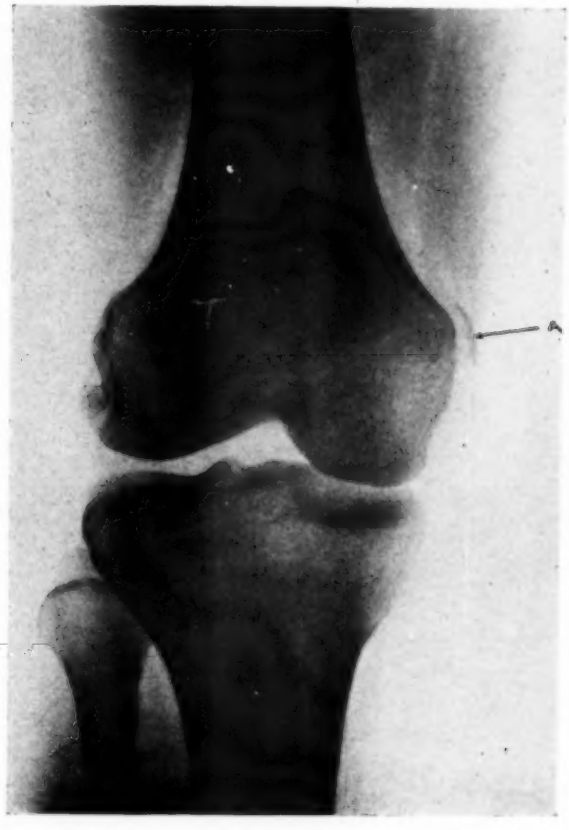


Fig. 2. Eight weeks after injury. Note calcification at A, just medial to superior portion of medial condyle of femur.

(Fig. 1.) Under local anesthesia a long No. 20 caliber needle was inserted into the knee joint and 80 c.c. of dark bloody fluid was aspirated. A wire splint was then applied from the toes to the mid-thigh and a pressure pad and bandage applied to the patella. The patient was discharged from the hospital on October 12, 1936.

Laboratory Reports.

Blood: White blood cells 6,700. Polymorphonuclears 77 per cent; lymphocytes 23 per cent.

Urinalysis: Negative.

Kahn: negative.

Progress Notes. 10-12-36. No swelling or tenderness over the left knee joint. Motion free. Muscle tone good. Splint replaced.

10-14-36. Small amount of fluid in joint. No tenderness. Motion free. Physiotherapy. Splint replaced.

10-16-36. Thirty c.c. of serosanguinous fluid removed from the knee joint. Motion free.

Slight tenderness over the medial condyle of the femur.

10-23-36. Fifty c.c. of serosanguinous fluid removed from the knee joint. Plaster cast applied from the toes to the anterior superior spine with the knee flexed 10 degrees and the foot in 5 degrees of plantar extension. The cast was bivalved to permit physiotherapy.

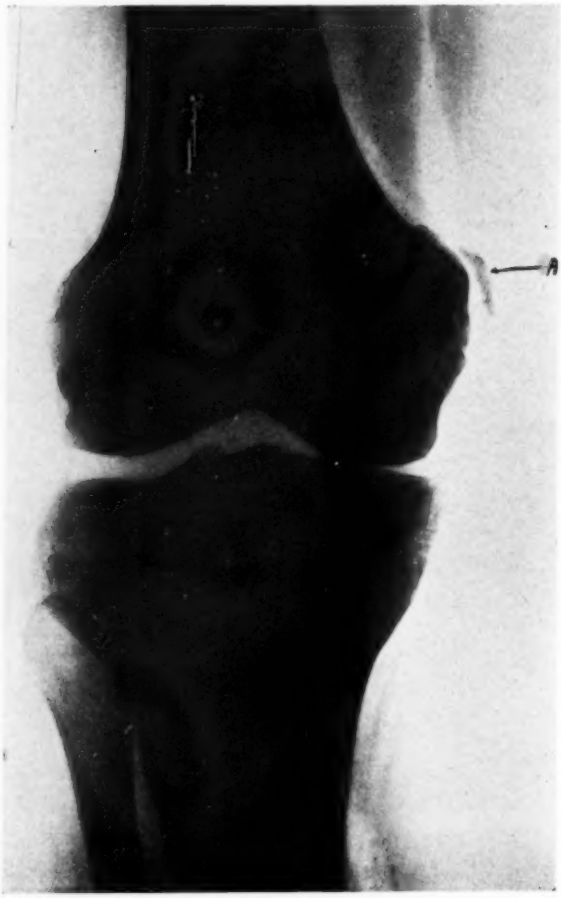


Fig. 3. Sixteen weeks following the trauma. Note increase in calcification at A. Patient able to work without difficulty.

11-6-36. Joint appears normal. No fluid. No tenderness. Motion free.

11-24-36. *X*-ray examination of the left knee joint revealed the following (Fig. 2): Examination of the left knee by films in the anteroposterior and lateral projections revealed no evidence of fracture, either old or recent. There was some calcium deposited in the soft tissues over the superior portion of the medial condyle of the femur—Pellegrini-Stieda's disease. The knee joint appeared normal, the articular surfaces being smooth, with the joint spacing preserved.

The patient was instructed to come to the office three times a week. At these visits he was given diathermy in the form of short wave (12 m) treatments using 2,500 milliamperes for twenty minutes. The fluid did not recur. Motion at the knee joint became free and painless. The joint was normal to palpation. He returned to work as a laborer and was discharged as cured on January 24, 1937. A check-up *x*-ray at this time showed that the calcification had increased somewhat in size as can be seen by comparing Figure 3, taken on this date, with Figure 2, taken in November 24, 1936.

COMMENT

A case of Pellegrini-Stieda's disease is reported in which the interval from the time of the accident to the formation of the characteristic bony lesion was eight weeks. An *x*-ray taken immediately following the accident showed no bony injury. This is one of the earliest appearances of the characteristic calcification on record. The knee was immobilized immediately when seen four days after the accident. The immobilization was continued for ten weeks. Even after the complete immobilization the calcification developed. Since the *x*-rays taken immediately following the accident showed no chip fracture, this case also refutes the earlier conception that this lesion is due to this type of fracture. We feel that the lesion is undoubtedly a metaplasia of connective tissue to form bone and that it ought not to be considered a separate entity from myositis ossificans. The definite space between the calcification and the femur and the outline and shape of the calcification makes the idea of a "fractureless callus" rather far fetched. (Fig. 3.)

Our treatment was conservative, the leg being completely immobilized. The patient returned to work fourteen weeks following the accident. Motion in the joint was free and painless. His only complaint upon discharge was that the joint became sore when the weather changed. Because the cases that were surgically treated by removal of the bony spicule recurred, and because of the excellent results we obtained by con-

servative treatment, we believe that this is the only feasible form of treatment for such cases.

SUMMARY

1. Pellegrini-Stieda's disease is merely myositis ossificans which happens to occur in the tibial collateral ligament because of trauma at that point.

2. Contrary to the impression given by the literature, the condition is quite common.

3. Treatment should be conservative for early return to work.

4. The presence of the calcification does not in itself constitute a disability, but should be considered as any other asymptomatic foreign body.

REFERENCES

1. Kulowski, J. Pellegrini-Stieda's disease. *J. A. M. A.*, 100: 1014 (April 1) 1933.
2. Riebel, F. A., and Riebel, J. A. Pellegrini-Stieda's disease. *Am. J. Surg.*, 24: 181 (April) 1934.
3. Oxford, T. M. Pellegrini-Stieda's disease. *Am. J. Surg.*, 27: 543 (March) 1935.
4. Wetzler, S. H., and Elconin, D. V. Calcification of the tibial collateral ligament. *Am. J. Surg.*, 27: 245 (Feb.) 1935.
5. Evans and Reynolds. Personal communication.

The sequence of events in bone necrosis appears to be: (1) septic embolism which cuts off the blood supply of a definite bone segment (infarct), and (2) subsequent invasion and infection of the infarcted area by bacteria from the embolus.
From—"Pediatric Surgery" by Edward C. Brenner (Lea & Febiger).

ACUTE INTESTINAL OBSTRUCTION COMPLICATING THYROTOXICOSIS*

ALBERT E. McEVERS, M.D.

Attending Staff, Hospital of the Good Samaritan, and the Cedars of Lebanon and Queen of Angels Hospitals, and Los Angeles County Hospital

LOS ANGELES, CALIFORNIA

ACUTE intestinal obstruction occurring in an individual already suffering from a severe hyperthyroidism presents a complicated surgical problem.

CASE REPORT

The patient, Mrs. V. L. L., age 24, white, mother of two children ages nine and seven years, was first seen at my office on January 7, 1937. She presented the clinical picture of severe hyperthyroidism: great muscular weakness, physical and mental restlessness, tremor, palpitation, tachycardia, flushing, sweating, loss of weight, blood pressure 160 systolic, 90 diastolic, prominence of eyes, with von Graefe's and Stellwag's signs positive. There was bronzing of the skin, a metabolic rate of plus 38, a cardiac murmur, but no fibrillation. The blood Wassermann was negative.

Past History. Appendectomy had been done ten years previous, tonsillectomy eight years previous, bilateral oöphorectomy, salpingectomy and supracervical amputation of the uterus five years previous when the patient was 19 years old. Following her surgical castration she had a severe menopausal syndrome, and also chronic obstruction of the bowels resulting from pelvic adhesions. She had several attacks of severe obstipation.

It was planned to send her to the hospital the first of the following week and start preoperative treatment for her hyperthyroidism. The day after her examination, about 6 P.M., she was seized with sudden cramp-like pains, described as being all over the abdomen, constant and severe in type. She vomited shortly after the onset and several times through the night. Two or three bowel movements occurred soon after the pain started, but none later. There was considerable rumbling in the bowels. The above history was given me the following morning when I was called.

Upon examining her I found tenderness across the lower abdomen, moderate distention, visible and audible peristalsis, pulse rate 180, marked palpitation, respirations rapid and shallow. The patient was in great pain and extremely nervous. The vomitus was not fecal in character. A diagnosis was made of acute intestinal obstruction complicating hyperthyroidism.

The patient was sent to the hospital and it was decided to operate immediately to relieve the obstruction. The usual preanesthesia medication had very little effect. Preoperative gastric lavage was not done. She was operated on at 2 P.M., twenty hours from the onset of the obstruction.

Operation. On January 9, under spinal anesthesia, an adequate incision was made. When the peritoneum was opened, approximately a pint of clear fluid escaped. The coils of the small bowel were distended and discolored. A quick exploration revealed the obstructive mass in the lower right quadrant. Several coils of bowel were fastened together with string adhesions; these were clipped one at a time and two or three loops of bowel were thus released. The obstruction was in the terminal ileum about 11 inches from its entrance into the cecum. This kink was found firmly attached to the pelvic floor and what was left of the right broad ligament by adhesions resulting from the previous surgery. Above the obstruction the bowel was distended; below, it was ribbon-like and flat and the colon was empty. The adhesions were separated and the kinked loop was delivered into the abdominal wound. The adhesions holding the kink together were cut and the bowel straightened out. Fecal material and gas readily passed through the previous point of obstruction into the empty bowel below. This kink of bowel, when released from its adhesions, presented a raw area approximately 10 × 4 cm., covered with granulating

* Presented at Staff meeting, Hospital of the Good Samaritan, February 8, 1937.

tissue. A free omental graft was sutured over this denuded area.

An enterostomy of the Witzel type was done.

The patient was placed in an oxygen tent and carbogen inhalations were given every two hours. A suction apparatus was used to clear

Fig. 1. Adhesions and obstruction in the terminal ileum. Insert: Free omental graft sutured over denuded area of bowel.

The catheter was anchored to the skin with silkworm-gut sutures. The abdomen was closed without other drainage.

Postoperative Treatment. Continuous subcutaneous and intravenous fluids were administered. Fifty minims of Lugol's solution were ordered daily. The enterostomy tube was connected to the Connell siphon apparatus; gas and material passed through the tube rapidly and drainage was most satisfactory. The next morning the patient had developed a thyroid crisis. She was irrational; her exophthalmos had increased markedly; the eyes remained open wide even in sleep, and the sclera and cornea became red and irritated; corneal ulceration occurred later. Temperature was 104°F., pulse rate about 180; there was a copious accumulation of mucus in the trachea. This whipped back and forth with the rapid respirations.

the throat of mucus. Intravenous injections of sodium iodide, gr. 15 were given daily instead of Lugol's solution; ice bags were applied to head, throat and heart; subcutaneous and intravenous fluids were continued. The fluid intake the first twenty-four hours after the operation totaled 6,000 c.c.—4,000 c.c. of sodium chloride solution by hypodermoclysis and 2,000 c.c. of 5 per cent glucose solution intravenously. Similar amounts were given for the next four days.

On the fifth postoperative day the patient developed a generalized edema. Subcutaneous and intravenous fluids were discontinued and fluids were given by mouth only, 2,900 c.c. on the fifth day and an adequate amount from then on daily. The edema soon subsided.

The patient was removed from the oxygen tent after seven days (January 17). The carbo-

gen inhalations were continued for two more days. Her thyrotoxicosis was under control on the eleventh postoperative day (January 20),

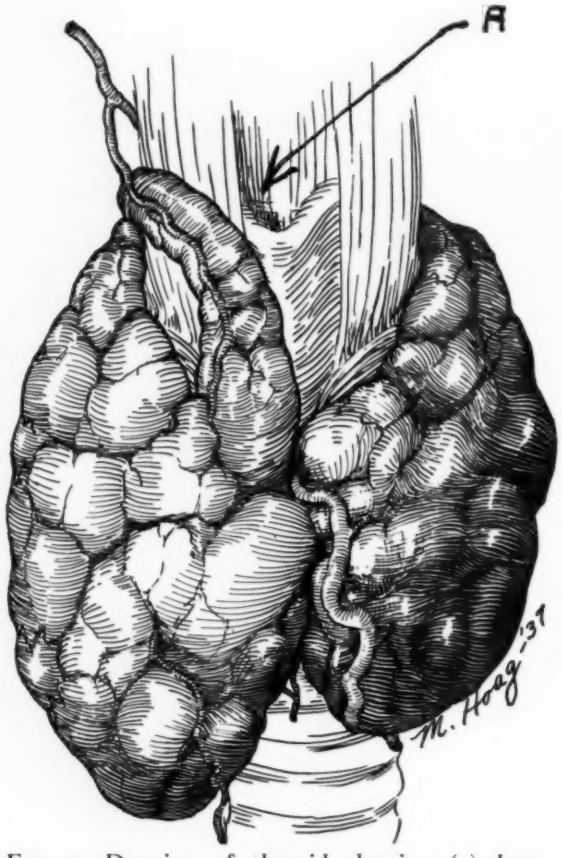


Fig. 2. Drawing of thyroid showing (*a*) large pyramidal lobe.

the pulse was 100, respirations 22, temperature normal. The enterostomy tube had drained well until this day, when it was removed. There was no subsequent drainage from the stab wound and no leakage around the tube while it was in place; the surrounding skin was not irritated.

For the next four days the patient remained about the same. On January 25, or the sixteenth postoperative day, she became more restless, hysterical at times, with a temperature of 102°F., a pulse of 140, and respirations 32. The pulse came down a little the following day but was still rapid; it was thought that she was getting out of iodine control. On January 27, or the eighteenth postoperative day the temperature was 103°F., pulse 140, respirations 28. On January 28, the temperature suddenly dropped to normal and pulse to 80. She remained in good condition January 29, and on the following day, the twenty-first after the bowel operation, she was taken to surgery and an adequate subtotal bilateral thyroidectomy was done.

Thyroidectomy. Under gas oxygen anes-

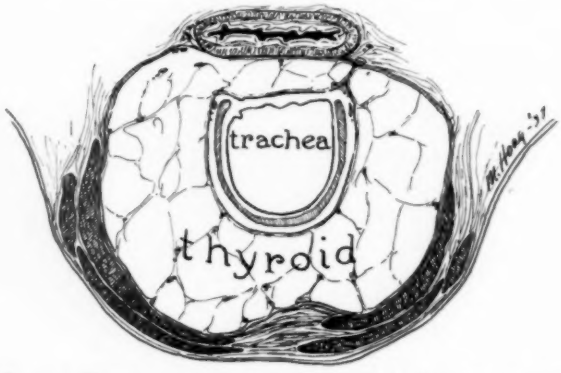


Fig. 3. Transverse section of thyroid showing superior pole right lobe in back of trachea and tracheal compression.

thesia the thyroid gland was exposed in the usual manner. A large pyramidal lobe was found situated to the right of the midline attached to the fascia covering the thyroid cartilage. Its base was connected with the isthmus and the apex extended up to the top on the right side of the thyroid cartilage. There was a good sized branch of the superior thyroid artery at its tip. The right upper pole extended around behind the trachea to its left side with resultant compression of the trachea.

Probably due to her recent severe tracheitis, the upper right superior pole was adherent to the trachea and esophagus. The thyroid tissue in the pole behind the trachea was about one-half inch thick. In order to visualize the tip and the superior thyroid vessels, it was necessary to displace the trachea to the left, and with a forcep on the upper end of the right lobe, make countertraction. By blunt dissection this pole was exposed so that the vessels could be doubly ligated and cut.

The internal capsule was adherent to the right lobe. However, a line of cleavage was found and the lateral and posterior aspect of the thyroid exposed. The inferior thyroid artery was visualized, the anterior branch ligated, and the inferior veins were ligated. There was an unusually large middle thyroid vein on the anterior surface of the isthmus running down to the left side of the trachea. The isthmus was separated from the trachea by blunt dissection, clamped and cut. The right lobe was resected, leaving a small piece of thyroid tissue about $2 \times 2 \times 4$ cm.

The patient being in fair condition, the left lobe was resected leaving a small piece of tissue approximating that which was left on the right side.

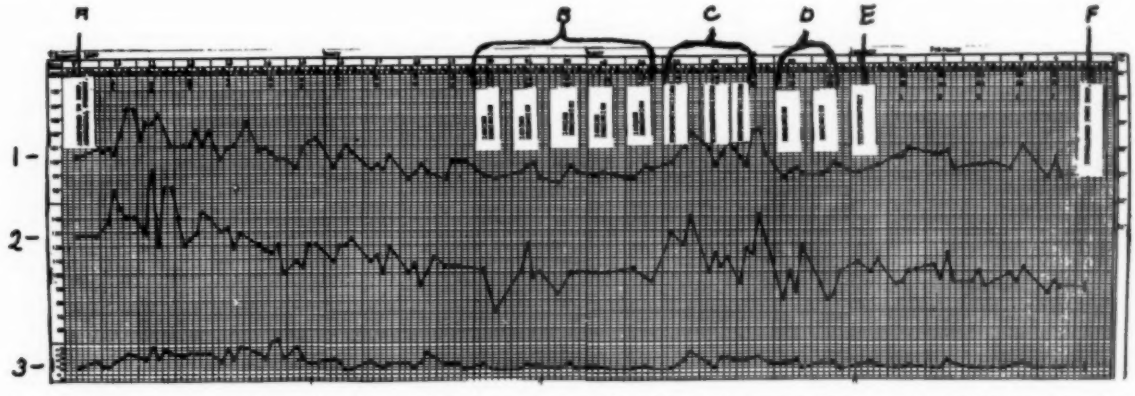


FIG. 4. Temperature chart showing: (1) temperature, (2) pulse, (3) respiration; *a*, admission to hospital and abdominal operation; *b*, remission the thirteenth, fourteenth, and fifteenth day of iodine administration; *c*, hyperthyroid symptoms sixteenth, seventeenth, and eighteenth day; *d*, nineteenth and twentieth day remission; *e*, thyroidectomy; *f*, discharged from hospital.

When the patient was returned to her room following thyroidectomy, she was immediately placed in the oxygen tent and given carbogen inhalations every two hours; continuous subcutaneous and intravenous fluids, sodium iodide intravenously, ice bags to head, throat and heart were continued and pantopon and nembutal were given in sufficient amounts to keep her drowsy. The total fluid intake the first twenty-four hours was 8,000 c.c.—5 000 c.c. of sodium chloride solution subcutaneously and 3,000 c.c. of 5 per cent glucose in sodium chloride solution intravenously. The second postoperative day she was removed from the oxygen tent, and additional fluids were given.

On the third postoperative day the skin clips were removed and she was allowed to sit up in a chair. The fourth and fifth days were uneventful; the wound was healed, dressings discontinued and the patient was up and about in good condition. There was no unfavorable reaction.

COMMENTS

1. The use of oxygen seemed well indicated. It not only dealt with the anoxemia associated with hyperthyroidism and tracheal compression, but also tended to overcome the anoxemia resulting from the use of morphine and the distention in the bowel. Dr. Fine[1] of Boston believes that if oxygen is administered for the distention associated with mechanical or functional obstruction, it has a very definite favorable benefit in aiding the elimination of gas from the intestinal lumen. Oschner[2] in a recent article agrees with Fine, and adds that part of the anoxemia associated with intestinal obstruction is probably due to the morphine usually administered.

2. The enormous amount of mucus that accumulated in the trachea was very uncomfortable and disturbing to the respiratory function. The mechanical suction apparatus with a catheter attached which was used to aspirate the mucus from the trachea was very satisfactory and gave immediate relief. The tracheal compression was increased by the swelling in the superior pole during the thyroid crisis. The patient was able to guide the catheter herself and the aspiration was repeated as frequently as the mucus accumulated.

3. The temperature chart shows that the peak of the remission occurred on the thirteenth day and remained there the fourteenth and fifteenth day. On the sixteenth day there was an exacerbation of the symptoms, suggesting that the patient was getting out of iodine control. However, by continuing with the iodine administration and other measures, remission was again obtained on the nineteenth day. This was maintained through the twentieth day and thyroidectomy was done on the twenty-first day.

4. Lahey[3] suggests that if a patient suffering with hyperthyroidism is operated upon within three weeks of a thyroid crisis one is rarely justified in performing a bilateral subtotal thyroidectomy in one stage. He recommends that the right lobe and the isthmus be resected in one stage and about six weeks later, the left lobe. In the case reported here it was felt that it would add very little additional risk to do a one-stage operation. Most of the pathology was in the right lobe and isthmus; the left lobe was small and its resection was quite simple.

REFERENCES

1. Fine, J., Sears, J. B., and Banks, B. M. The effect of oxygen inhalation on gaseous distention of the stomach and small intestine. *Am. J. Digest. Dis. & Nutrition* (Aug.) 1935.
2. Oschner, Alton, and Storck, Abmrose H. Mechanical decompression of the small intestine in ileus. *J. A. M. A.*, 108: 260–266 (Jan. 23) 1937.
3. Lahey, Frank H. Critical thyroid states, their diagnosis and treatment. *Surg. Clin. North America*, 16: 1521–1531 (Dec.) 1936.

Continued external biliary fistula is always marked by a profound progressive anemia, diminution of hemoglobin and red blood cells.
From—"Bile—Its Toxicity and Relation to Disease" by O. H. Horrall (University of Chicago).

NEW INSTRUMENTS

SUCTION APPARATUS*

VICTOR CARABBA, M.D., F.A.C.S.

NEW YORK CITY

THE value of suction as a therapeutic procedure in surgery, has been definitely established. The great contribution of Wangensteen[1] in the use of suction in the relief of intestinal distention is a noteworthy example. The other surgical conditions in which suction can be employed are: stomach drainage, duodenal drainage, biliary drainage, and suprapubic drainage.

All of the apparatus used since 1917 for slow suction in surgical conditions have been vacuums produced by water siphons. Mechling,[2] in 1917, described the earliest suction apparatus. The apparatus consisted of tanks revolubly supported on a frame and arranged one above the other with a means of permitting the fluid of one tank to pass to the other tank to cause a vacuum in one of the tanks. Felsen,[3] in 1918, described a suction apparatus for thoracentesis. The apparatus consisted of a hermetically closable liquid reservoir, adapted to contain a partial filling of liquid and an air space above the level of the fluid. A vent in the bottom of the bottle permitted gravity drainage of the bottle with resulting suction produced in the air space above. Tuttle,[4] in 1919, patented an apparatus which used the same principle as that of Felsen, but was larger and more complicated in its detailed mechanism. Bethune,[5] in 1921, patented a reversible double container vacuum pump so connected that, when fluid passed from the upper to the lower container, a vacuum was produced above the level of the liquid in the upper container. Gaynor and Wheelwright,[6] in 1935, reinvented the apparatus described by Bethune, adding a more elaborate valve mechanism. Wangensteen earlier described his simple water siphon suction device.

One of the principal objects of the present apparatus is to provide an improved vacuum siphon apparatus, wherein air, rather than water, is employed for the purpose of producing the relatively low vacuum, which is necessary to provide a proper order of drainage.

Another object is to suggest an improved suction apparatus which is small in size and readily movable, light in weight and easily operated. The device will function for a considerable period of time without any attention on the part of the operator, and is readily controllable as to the amount of fluid which is drawn from the body.

The suction device comprises a collection bottle of suitable size, the bottle having a pair of flexible ducts leading therefrom, one of such ducts leading to the patient and the other to a somewhat larger bottle, preferably spherical in shape and containing an inflatable bag. A tube leads into the bag, the opposite end of the tube having means for connection with a conventional air pump. The tube also has a plurality of valves or petcocks in the line of flow of fluid passing through it. One of the petcocks is of conventional form and the other is formed with an aperture in the valve, so that a slight flow of fluid may take place even when it is closed. This

* From New York University College of Medicine and Third Surgical Division Bellevue Hospital, Arthur M. Wright, M.D., Director.

aperture should be large enough to permit about 30 c.c. of air to pass through per minute.

naturally ceases, due to the equalization of pressures. If the duct from the collection bottle is to be left in the patient afterward,

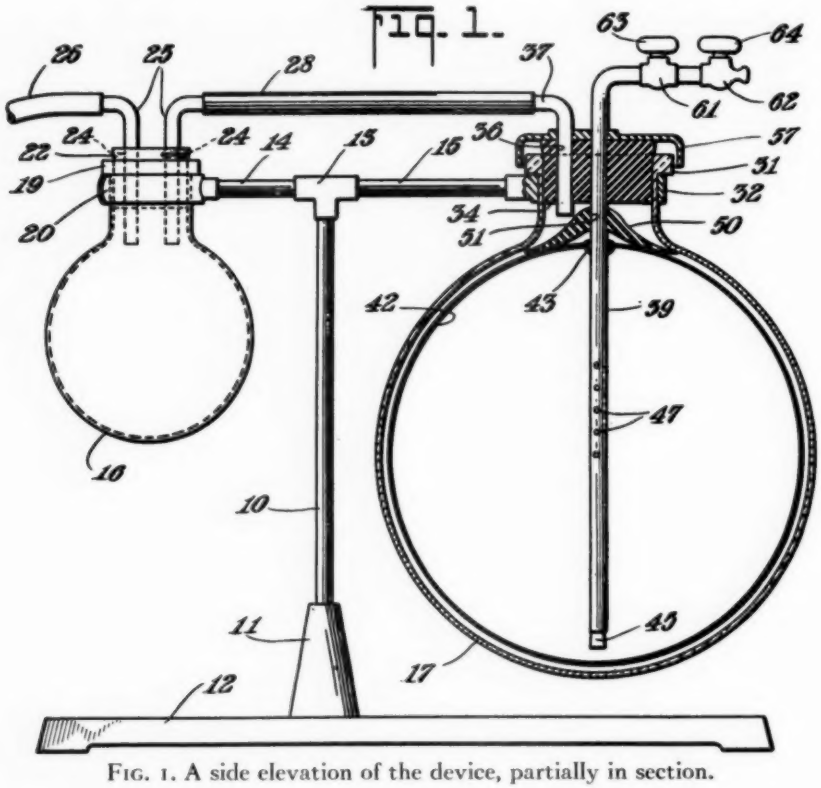


Fig. 1. A side elevation of the device, partially in section.

The bag (Fig. 2) is inflated by a conventional pump to a point where it more or less completely fills the spherical vessel, after which both valves are closed. When the device (Fig. 3) is to be used, the first mentioned valve is opened, thereby permitting 30 c.c. of air to pass out of the bag per minute. In so doing, a partial vacuum is created in the larger vessel which is connected to the collection bottle by a flexible duct. Within three or four minutes, the air in the collection bottle is withdrawn to a point wherein the suction becomes effective and fluid is withdrawn from the patient.

The suction continues as long as there is fluid in the cavity of the patient, or as long as there is air in the cavity at a pressure in excess of atmospheric pressure. When the air in excess of atmospheric pressure, and the fluid have been withdrawn from the cavity, the suction quite no further air will pass from the inflated bag until the suction again begins to function.

The device (Fig. 1) is mounted on a vertical support (10), which is made from tubing of suitable diameter to provide the desired strength, in a socket (11) at its lower end, the socket in turn being secured to an elongated substantially rectangular base (12). A "T" fitting (13) is mounted at the upper end of the vertical support (10). The "T" carries horizontal tubular members 14 and 15, the tube (14) supporting the relatively smaller collection bottle (16), and the tubing (15) supporting the larger, spherical, vacuum-producing bottle (17).

The upper end of the collection bottle is formed with the usual flange (19) which is supported by a substantially U-shaped collar (20) carried by the tube (14). A stopper (22), made of rubber or other

suitable material, is placed in the upper end of the collection bottle; the stopper is formed with a pair of spaced longitudinal flange (31) at its upper end which rests upon a circular collar (32) carried by the horizontal tube (15).

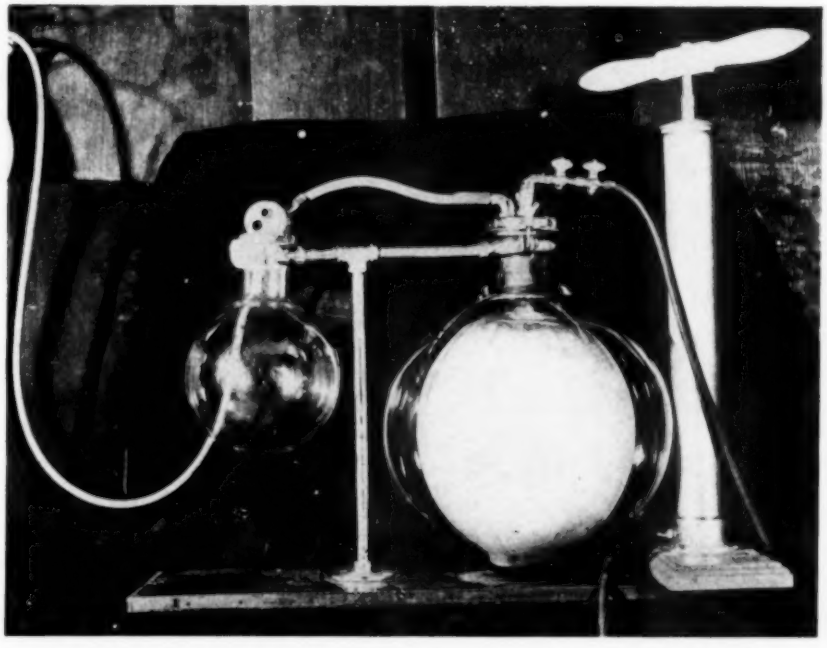


Fig. 2. Bag being inflated, stopper disconnected in collection bottle.

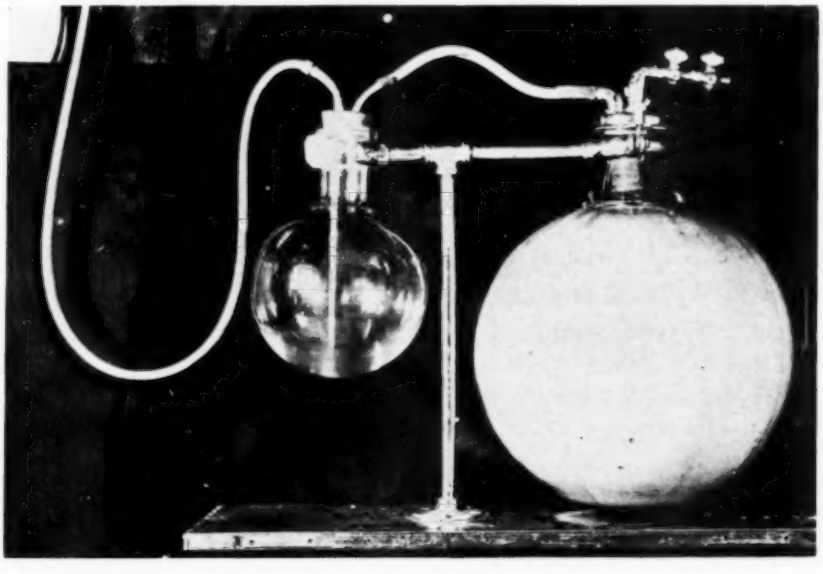


Fig. 3. Stopper in collection bottle replaced. Petcocks open, air escaping from bag to produce vacuum.

apertures (24), which contain inlet tubes (25). To one of these tubes is secured a length of rubber tubing (26) which passes to the patient; the other tube carries a shorter length of rubber tubing (28) which passes to the vacuum-producing bottle (17). This bottle is likewise provided with a

A rubber stopper (34) within the opening of the bottle (17) is provided with a pair of spaced apertures (36). In one of these there is a short inlet tube (37) connected with the rubber tube (28) and extending into the bottle (17) only a short distance below the stopper (34). The second

aperture in the stopper holds a somewhat longer tube (39) which extends downward to a point adjacent to the lower end of the bottle.

An inflatable rubber bag (42), having a reinforced opening (43), is mounted on the tube, the tube passing through it in such a way that an air-tight seal is formed. The bag is of such size, that when inflated it completely fills the spherical bottle (17) in which it is contained. In order to prevent injury to the bag, a suitable stopper (45) is placed in the aperture at the lower end of the tube. The tube also has several spaced apertures (47) in its side walls, so that air may pass from the tube into the bag.

Just above the bag there is an annular gasket (50) with a central aperture (51) through which the tube (39) passes. The gasket is of slightly greater diameter than the diameter of the neck of the bottle in order to prevent the inflated bag from coming in contact with the lower end of the tube (37). The collar (32) which supports the bottle (17) is provided with upward extending bolts (54) on opposite sides, and the bolts are provided with wing nuts (55). There is a metal cap (57) with a pair of spaced ears (58) on opposite sides at the upper end of the bottle above the stopper (34). The screws (54) pass between these spaced ears; the cap (57) is held in firm engagement with the upper end of the bottle by means of the wing nuts (55). The upper end of the tube (39) carries a pair of petcocks (61) and (62), provided with valve handles (63) and (64). One petcock (62) is of the conventional type and the other (61) is provided with a valve (not shown) which, when closed, permits a small quantity of air, about 30 c.c. to pass through per minute.

To inflate the bag, the petcocks are opened and a conventional pump with check-valve (not shown) is attached to the nipple (65) on the valve (62). When the bag is completely inflated, the valves are closed, the pump removed, and the device is ready for use.

In use, the flexible tube is first connected with the patient and the valve in the petcock 62 opened, thereby permitting approximately 30 c.c. of air to pass out of the bag per minute. In four minutes or thereabouts, a sufficient quantity of the air is withdrawn from the collection bottle (16), to cause the suction apparatus to become effective and start the withdrawal of fluid from the patient.

When the fluid in the patient's cavity has for the time being become exhausted, or when the air pressure within the cavity has been reduced to atmospheric pressure, the suction ceases by the equalization of the pressures within and without the device. It is not necessary, however, to shut the valve (62) at this time, since suction again begins to function as soon as more fluid has collected in the cavity.

REFERENCES

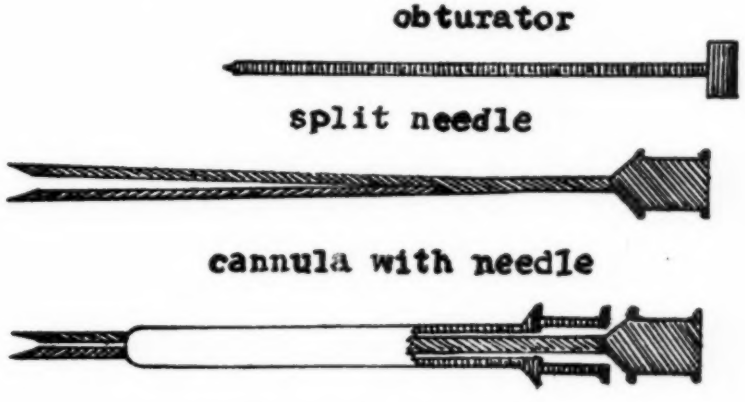
1. Wangensteen, O. H. Rationalizing treatment in acute intestinal obstruction. *Surg., Gynec. & Obst.*, 64 (Feb. 15) 1937.
2. Mechling, T. F. U. S. Patent 1,250,928. Vacuum-Siphon.
3. Felsen, J. Apparatus for thoracentesis. U. S. Patent 1,284,630.
4. Tuttle, A. H. Suction apparatus for surgical use. U. S. Patent 1,316,340.
5. Bethune, C. W. Suction pump. U. S. Patent 1,374,808.
6. Gaynor, W. C. T., and Wheelwright. New suction apparatus. *Am. J. Surg.*, 30: 185 (Oct.) 1935.

A NEW BIOPSY NEEDLE

IRVING SILVERMAN, M.D.

BROOKLYN, NEW YORK

TODAY so much emphasis is being placed on the early diagnosis of cancer that the utmost importance is attached to biopsy instruments designed to yield the maximum amount of tissue with a minimum amount of trauma. Hitherto the aspiration biopsy has been considered the simplest and least traumatizing method and has been used in many cancer clinics throughout the country. The practical value of a positive biopsy has greatly outweighed any theoretical objections to its use. Briefly, the method consists of inserting a needle of about 17 gauge into a suspected tumor, aspirating the material and then spreading it out between two glass slides. In these smears enough tumor cells may be found to make a correct diagnosis possible. Unfortunately, the percentage of positive smears has proved to be extremely variable, and for this reason many modifications have been devised to insure the extraction of tissue. These consist of wires or metallic devices with corkscrew tips, barbs, or dental burrs, fused to the ends, and introduced into the lumen of the aspirating needle. Somewhat larger instruments were devised by Muir, Hoffman, Lindblom, and Corriero.



A NEW BIOPSY NEEDLE

FIG. 1.

FIG. 2.

The biopsy needle which is now presented was devised in the tumor clinic of the Caledonian Hospital on the service of Dr. Joseph Tenopyr. It is believed to be superior to all others heretofore used for the same purpose because it causes as little trauma as a simple aspiration biopsy, takes only a few moments to do, and yields a larger piece of tissue in proportion to its size. With a 16 gauge needle a piece of tissue 2 by 10 mm. is obtained. A single punc-

ture is sufficient to obtain tissue, and repeated insertion of the needle is not necessary.

The instrument consists of an outer needle or cannula of 16 gauge, and an inner needle of 18 gauge which is split longitudinally down to the hub. To obtain a specimen, the skin over the suspected mass is prepared with tincture of iodine and alcohol, and then 1 c.c. of a solution of ½ per cent procaine hydrochloride is injected intradermally and subcutaneously over the site of puncture. The outer needle or cannula, with obturator in place, is inserted into the tissue up to the suspected tumor. The obturator is then removed and the split needle inserted in its place and pushed into the tumor mass cutting a cylinder of tissue between its prongs. The outer needle or cannula is now advanced over the split needle into the tumor for a distance of about ½ inch. The entire instrument is then rotated slightly, after which the inner needle is withdrawn with the specimen. The latter is then placed in 10 per cent formalin and prepared for cutting in paraffin and staining in the usual manner.

In the absence of bacteriemia [in acute hematogenous osteomyelitis] and in the average case accompanied by mild infection, operation is contraindicated *unless suppuration develops:* within the bone, beneath the periosteum, or in the soft parts.
From—"Pediatric Surgery" by Edward C. Brenner (Lea & Febiger).

[From Fernelius' Universa Medicina, Geneva, 1679.]

BOOKSHELF BROWSING

PREDISPOSITION TO CANCER IN THE BONAPARTE FAMILY

BORIS SOKOLOFF, M.D.

NEW YORK CITY

VERY few are aware of the fact that throughout his life Napoleon Bonaparte was obsessed by secret fear of the "familial disease." While he refrained, almost to the point of superstition, from discussing this subject, yet certain phrases dropped here and there, and duly recorded by witnesses, permit us to believe that from the day he learned of his father's death, the thought that he might fall heir to the same disease never left him.

In 1806, when he suffered a mild attack of gastralgia, he remarked to Count Lobau that he "bore in himself the germ of a premature death."[1] Evidently as time went on he became even more seriously concerned. In 1811, he ordered that a copy of his father's autopsy report be obtained and then studied it carefully. Shortly afterwards, he suddenly manifested an interest in anatomy and even began to take lessons from his court physician, Dr. Corvisart, although in absolute secrecy. Instruction was illustrated by portions of cadavers brought into the palace wrapped in towels. Dr. Corvisart recollects that the human stomach was an object of Napoleon's special interest, but the lessons were soon discontinued, as the Emperor could not bear the sight of dead flesh. His knowledge of anatomy apparently did not progress to any extent, for years later while an exile on St. Helena and seriously ill, he once more questioned his attending physician, Dr. Arnott, at great length on the anatomy of the human stomach. On hearing the term "pylorus," he asserted quite gravely that there lay the seat of his illness and added "I am dying from the same disease as my father."[2] In delirium and near death, he kept repeating "Pylorus . . . my father's pylorus."[3]

Some of his brothers and sisters, as well, lived under the hypnosis of the same idea—that they were doomed to die from cancer.

The Bonapartes' predisposition to cancer has been referred to more than once by Napoleon's biographers. However, this question has never been fully discussed in medical journals. One of the most noted of Napoleon's biographers, Frederic Masson, remarked in this connection:

"The Bonapartes had no hereditary flaw. They appear to have been disposed, more than others, to an illness which is claimed not to be hereditary—cancer. Several of them succumbed to it, but the majority escaped. Four cases out of nearly forty do not constitute a family flaw."[4]

However, it would undoubtedly constitute a predisposition to cancer if four members of the family died from this

disease. In the present paper, the case histories of some of the Bonapartes are briefly summarized, based on available medical records, and the predisposition of the Bonapartes to cancer is analyzed in the light of our present-day medical knowledge.

CASE HISTORY: CHARLES BONAPARTE, NAPOLEON'S FATHER

Little is known of Charles Bonaparte's parents, except that his father died at the age of 40 from stomach disease. There is some indication of cancer. No information is available concerning his mother.

The first symptoms of gastric dysfunction became evident in 1783 when Charles Bonaparte was 37. He complained of frequent vomiting, loss of weight and pain in the epigastric region. In November 1784, he consulted Dr. Tournatoire at Aix, whose diagnosis was advanced carcinoma of the stomach. Bonaparte left for Montpellier, where he was treated by Drs. Farjon and Lamure, who agreed that he was suffering from a large tumor in the pyloric portion of the stomach.[5] He died February 24, 1785, at the age of 39. An autopsy was performed at the Montpellier Medical School by Dr. Bousquet, Surgeon-in-Chief, assisted by Dr. Fabre, an intern in the Department of Surgery. The post-mortem findings were as follows:

"The stomach was found to be distended and filled with the liquid that the patient has ingested; the inferior orifice of the organ formed a tumor of semicartilaginous consistency, which was of the shape and size of a large potato or a large elongated pear. The lining of the stomach towards its greater curvature was very much thickened and of hard consistency, like that of cartilage; the thickness of the lining was increased nearing the pylorus, and the inferior orifice of the stomach was enclosed in the center of the tumor, and was found to be so contracted that one had to use a scalpel in order to introduce the tip of the finger; this tumor did not extend beyond the pylorus. The duodenum was in its natural state. The liver was found to be partially congested and the gall-bladder, completely filled with very dark bile, had acquired the volume of a medium sized elongated pear. Although the pancreas seemed very sound, granules of a larger size than is natural were easily perceived."[6]

Although a microscopic examination was not made, there is little doubt that Charles Bonaparte, father of Napoleon, died of carcinoma, the tumor being of a scirrhous type. The post-mortem diagnosis confirmed the one made by his physician a year before his death.

CASE HISTORY: NAPOLEON BONAPARTE

His father died from cancer of the stomach at the age of 39. There is indication that his grandfather, Joseph Bonaparte, also suffered from the same disease. His mother, Letizia, died of pneumonia at 70. As a child, Napoleon suffered from jaundice and intestinal disturbance. At the age of 18, he had an attack of malarial fever (Dr. Bienvelot). At the age of 23, he contracted a scabious skin infection (Sarcoptes), from which he continued to suffer for more than eight years. Gradually, chronic eczema developed. At the age of 25, he experienced a grave intestinal infection (Dr. Desgenettes). From that time on, he complained of chronic constipation and hemorrhoids. He had a temporary attack of jaundice (acholuric icterus). From about 28 to 30, he suffered from a persistent cough accompanied by fever; on one occasion, hemoptysis of pulmonary origin was manifested (Dr. Corvisart). About that time, he began to suffer from dysuria, due to non-gonococcic urethritis (Dr. Boyer, Dr. Ivan).

After 30, gastric symptoms gradually increased in frequency, indicating the presence of chronic gastritis. There was persistent constipation. At the age of 47, at St. Helena, a scorbutic condition developed, accompanied by tonsillitis, hypoglossitis and swelling of the gums (Dr. O'Meara). In July 1817, at the age of 48, he had an attack of bronchial catarrh. In September 1817, he suffered from edema of the feet, nausea, a dull sensation in the right hypochondriac area and numbness in

the right scapular region. The diagnosis of Drs. O'Meara and Stokoe was acute hepatitis. The symptoms of dyspepsia increased—nausea, flatulence, vomiting. There was no pain in the gastric region, however, but a dull sensation still persisted in the right hypochondriac area. Anorexia and melancholy were pronounced.

In 1819, there was marked anemia accompanied by accentuated jaundice. A diagnosis of chronic hepatitis was made by Dr. Antommarchi. In July 1820, fever, bilious vomiting and persistent diarrhea began. On August 17, 1820, he complained for the first time of a knife-like pain in the right hypochondrium and hypochrosis and acroparasthesia were observed. From then on, his condition gradually grew worse. His stomach rejected all food; he was confined to bed. Yet, his physicians were still optimistic. Dr. Antommarchi's diagnosis in March 1821 was "gastrica pituitosa fever" (acute gastritis). On April 2, 1821, Dr. Arnott diagnosed the case as gastritis, hypochondriasis and secondary anemia. The consultation of Drs. Shortt, Mitchell, Arnott and Antommarchi resulted in no definite diagnosis. On April 23, the diagnosis of dyspepsia and hypochondriasis was upheld by Dr. Arnott. For the first time, on April 25, the presence of gastric ulceration was suggested by Arnott. Constant vomiting, fever, and delirium were present. Napoleon died at 5:49 on the afternoon of May 5, 1821.[7] On May 6, an autopsy was performed by Drs. Shortt, Mitchell, Livingstone, Arnott, Rutledge, Henry and Antommarchi.

Autopsy Findings. "On the anterior surface of the stomach, near the small curve, at a distance of about three fingers from the pylorus, there was a slight obstruction, apparently of a scirrhous nature, and distinctly circumscribed. The stomach wall was perforated through and through and in the center of that small induration, the aperture of which was closed by adhesion of that part to the left lobe of the liver.

"On opening the stomach along its greater curvature, it was observed that the mucous membrane of the stomach was sound from the lesser to the greater cavity, following the greater curvature. Almost the whole of the remainder of the internal surface of the stomach was occupied by a cancerous ulcer, the center of which was in the upper portion along the lesser curvature of the stomach, while the irregular digital and linguiform border of its circumference extended both before and behind that internal surface and from the orifice of the cardia, to within a good inch of the pylorus. Its rounded opening scarcely occupied a diameter of four or five lines inside and at the most, two and a half lines outside. The circular border of that opening was extremely thin, slightly denticulated, blackish and formed only by the peritoneal membrane of the stomach. An ulcerous, grayish and smooth surface lined this canal, which, but for the adhesion of the liver, would have established a communication between the cavity of the stomach and that of the abdomen. The right extremity of the stomach, at a distance of one inch from the pylorus, was surrounded by a tumor, or rather a scirrhous annular induration, a few lines in width. The orifice of the pylorus was in a perfect condition. The lips of the ulcer exhibited a remarkable fungus-like swelling, the bases of which were hard, thick and in a scirrhous state."[1]

The physicians present at the autopsy all agreed that it was cancer in the pyloric region of the stomach that caused Napoleon's death.

However, no microscopic examination has ever been made, since the stomach tissue was not preserved. Therefore, at best, a diagnosis can be only speculative, based on the macroscopic description. It may have been either adenocarcinoma of the stomach or a benign callous ulcer of long duration. But the clinical records and the physicians' daily reports strongly suggest a malignancy of the stomach. The absence of gastrorrhagia and postprandial pain during the three years' course of the disease do not support the hypothesis of the presence of a benign ulcer of long duration, which may have gradually degenerated into cancer. The presence of an ulcer is usually accompanied by symptoms which

were absent in Napoleon's case. He experienced a knife-like pain for the first time only a few months before his death. Could a physicians, but who was not present at the autopsy, having left the island of St. Helena two years prior to Napoleon's

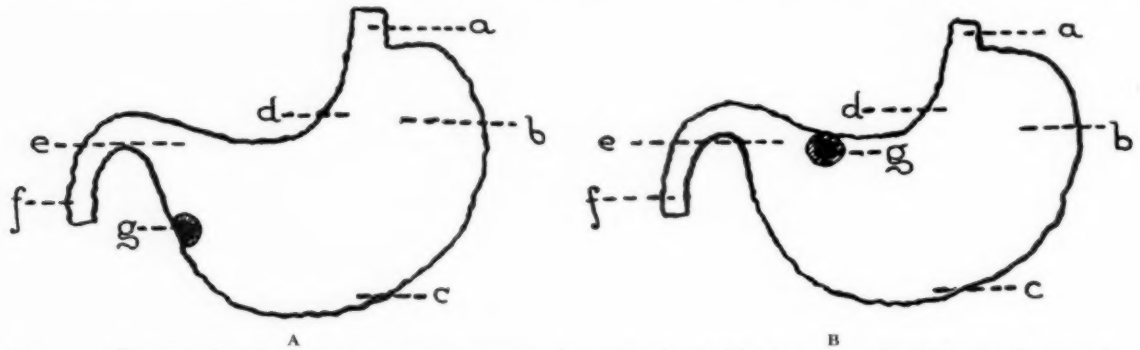


Fig. 1. A, diagram showing the approximate center of tumefaction (g) in the stomach of Charles Bonaparte, Napoleon's father. B, approximate center of tumefaction in the stomach of Napoleon Bonaparte. *a*, esophagus; *b*, cardiac end of stomach; *c*, greater curvature; *d*, lesser curvature; *e*, pylorus; *f*, duodenum.

benign ulcer have progressed so rapidly in eight months so that practically two-thirds of the stomach was affected? We do not think so. The whole history of Napoleon's last illness indicates a malignancy of the lesser curvature of the stomach—probably an infiltrating adenocarcinoma which gradually became ulcerated. This diagnosis is confirmed by the general condition of the patient, which was typical of cases of malignancy. The fact remains, however, that a definite diagnosis was made only after his death, although he had been attended by several physicians.

The following diagrams show the location of the tumors in the stomachs of Charles and Napoleon Bonaparte, according to the autopsy findings. (Fig. 1.)

Some years ago (1912–1913) there was a discussion in the British Medical Journal concerning the condition of Napoleon's intestines. The fact is that in the British Museum of the Royal College of Surgeons, there are two fragments of intestines labelled, "Incipient Fungus in the Glands of the Intestines of Napoleon." The authenticity of these pieces is very doubtful, according to Dr. Arnold Chaplin.[8]

Sir Astley Paston Cooper,[9] the famous pathologist of the last century, was the donor of these fragments to the museum. It seems that he had received them from Dr. O'Meara, who was one of Napoleon's death. Sir Astley Cooper, judging from the histologic sections made of these fragments, considered that they showed cancerous metastasis of the intestines. If this is so, then one should presume that cancer of the stomach was of long standing and that it originated much earlier than the clinical symptoms. However, another histologic survey, made a hundred years later by Dr. A. Keith,[10] showed that these pieces did not at all present a picture of cancerous metastasis, even if they were authentic. Keith himself thinks that the fragments present a lymphoid hyperplasia due to attacks of malaria. Outside of the fact that the authenticity of these fragments is more than doubtful, Keith's investigation has no connection with the primary malignancy of Napoleon.

As autopsies on the bodies of the other members of the Bonaparte family were not performed, only clinical observations concerning them are available. An exception is Napoleon's son, the young Duke of Reichstadt, who died at the age of twenty-one from acute pulmonary tuberculosis (Dr. Malfati).

Elisa, Napoleon's eldest sister, suffered from dyspepsia from youth. A diagnosis of hypochondriasis and gastritis was made by Dr. Barthez of Carcassonne and Professor Touquet of Montpellier. At the age of 40, there was a pronounced hematopedesis,

gastric symptoms, nausea and frequent vomiting, and a continuous fever. She died at the age of 43. Gastric cancer was suspected.

Pauline, Napoleon's second sister, complained of gastric symptoms for years. During the last three years of her life she refused all food. She died at the age of 44. Physicians suspected that the cause was phthisis and cancer of the stomach.

Caroline, Napoleon's youngest sister during the last two years of her life suffered from jaundice and stubborn vomiting. Two lesions were found, one in the stomach and the other in the colon, the latter appearing to be secondary. The diagnosis, made by her physician Dr. Playfair, was carcinoma of the stomach.

Joseph, Napoleon's eldest brother, suffered from nephritis (Dr. Dubois). There were signs of a gastric disturbance. He died from cerebral hemorrhage.

Lucien, Napoleon's second brother, during the last years of his life, complained of pain and gastric symptoms—nausea and vomiting, accompanied by icterus. He died at the age of 65. Cancer of the stomach was suspected.

Louis, Napoleon's third brother, died of apoplexy at the age of 68. He complained at times of gastrointestinal disturbance. His son, Napoleon III, died in 1873 of pyelonephritis.

Jerome, Napoleon's youngest brother, was diabetic and died of pneumonia at the age of 76.

The following table summarizes the data concerning these members of the Bonaparte family:

JOSEPH BONAPARTE (Died at the age of 40. Cancer of the stomach suspected.)

CHARLES BONAPARTE (Died at the age of 39 of scirrhous carcinoma of the stomach.)

ELISA	PAULINE	CAROLINE	NAPOLEON BONAPARTE	JOSEPH	LUCIEN	LOUIS	JEROME
(Died at the age of 49. Cancer of stomach suspected.)	(Died at the age of 44. Cancer of the stomach suspected.)	(Died at the age of 56. Cancer of the stomach.)	(Died at the age of 52. Adenocarcinoma (?) of stomach.)	(Died of cerebral hemorrhage.)	(Died at the age of 65. Cancer of stomach suspected.)	(Died at the age of 68 of apoplexy.)	(Died at the age of 76 of pneumonia.)

NAPOLEON II. Duke of Reichstadt. (Died of pulmonary tbc. at the age of 21.)

Although only three members of Napoleon's family unquestionably suffered from malignancy of the stomach—his father, himself and his sister, Caroline—four other members are suspected to have been victims of cancer. In general, the majority of Napoleon's generation suffered from gastric dysfunction and manifested from their youth symptoms of dyspepsia and gastritis. However, the following generations of this family were completely free from incidence of malignancy.

DISCUSSION

Bonaparte's family presents an interesting case of familial predisposition to cancer, particularly because the malignancy originated in the stomach. The recent work by Loeb, Lacassagne and others has somewhat clarified the obscure question of cancer heredity.[11,12] From their extensive laboratory experiments, it seems evident that there does not exist such a thing as the simple transmission of cancer by inheritance. According to these authors, the difficulty of forming a line of non-cancerous homozygotes is not compatible with the

idea that cancer depends upon a simple Mendelian factor. Therefore, the theory defended by Maud Slye did not receive support in these more recent experiments on this subject. Nevertheless, the inherited predisposition to malignancy is still considered a prime factor in the occurrence of cancer in animals. The hormonal pathogenesis of adenocarcinoma of the breast in mice has given conclusive evidence in this respect. From these experiments, we may see that mice from a low incidence line of cancer are resistant to heavy doses of carcinogenic hormonal substances, such as estrin. And again, mice of pure strain with a high incidence of cancer are extremely sensitive, even to relatively small doses of estrin. Their predisposition to cancer is such that not only the female, but even the male, may develop cancer of the breast after application of estrin.

Leo Loeb demonstrated that incidence of the breast in mice of a pure strain can be considerably increased by carcinogenic hormonal substance. But as soon as the purity of the line is broken by heretozygotis, this predisposition to cancer is reduced, or even completely annihilated. The nature of predisposition to malignancy has not yet been determined, but it may have some relation to glandular physiology. We know that large doses of estrin produce hyperplasia of the glandular system, particularly of the anterior pituitary and adrenal cortex (Burrow, Cramer[13]).

Referring again to the Bonaparte family, we may note that cancer occurred only in the first two generations. In the next two generations, the predisposition to cancer seems to have been broken by heterozygotis. Therefore, Masson's statement needs substantial corroboration. Among the eleven members of the older generation of Bonapartes, three were undoubtedly afflicted with cancer and four of them were suspected cases of malignancy; but among the thirty members of the younger generation, not one suffered from this disease. Thus, the predisposition to cancer in the Bonaparte family was pronounced in practically but one generation, that of Napoleon.

An analysis of Bonaparte's case may convince one that *human predisposition to cancer is a phenomenon of an unstable, variable nature, which is easily broken by marriage.* It is therefore of doubtful practical value in the prognosis of malignancy. Although familial predisposition should be taken into consideration, its importance must not be overestimated. However, a study of families with cancer incidence is of prime importance for our better knowledge of cancer predisposition among humans. An analysis of the Bonaparte family seems to be in complete accord with recent experimental data.

I wish to thank Dr. Leo Loeb, head of the Department of Pathology of Washington University Medical School for his valuable suggestion in connection with Napoleon's case.

REFERENCES

1. Segur. Mémoires, vol. iv, p. 82, Paris.
2. General Bertrand. Campagne d'Égypte et de Syrie, vol. ii.
3. Antommarchi. The Last Days of Napoleon. London, 1826. H. Colburn Co.
4. Masson, Fred. Napoleon and His Son. Paris.
5. Grasset-Morel, Les Bonapartes à Montpelier. 1900. Also Dr. Cabanes. Au chevet de l'Empereur. Paris, 1920. Albert Michel.
6. "Certified identical with the original, remaining in my hands at the disposal of authorities"; Signed, Baron A. Dubois (m.d.) Paris, July 12, 1811.
7. Reference to Napoleon's illness: See Napoleon's Medical Biography, by Boris Sokoloff. New York, 1937. Prentice Hall Co.
8. Chaplin, Arnold. Napoleon's Illness. London, 1913. Hirschfeld Bro.
9. Cooper, Sir Astley Paston. Met Dr. O'Meara in 1819 (1768–1841).
10. Keith, A. *Brit. M. J.*, p. 183–238, 1913.
11. Lacassagne. *Am. J. Cancer*, 27: 217, 1936.
12. Loeb, Leo. *Am. J. Cancer*, 27: 229, 1936.
13. Cramer and Horning. *Lancet*, 1: 247, 1937.

AUTHOR INDEX TO VOLUME XL

Adelstein, Leo J., 384
Albright, Hollis, 282
Allen, Arthur W., 35
Allen, Clyde I., 89
Andrews, V. L., 454

Balensweig, Irvin, 654
Baumeister, Carl, 626
Baumgarten, Elden C., 658
Benedict, Edward B., 5
Benmosche, M., 618
Benson, Clifford D., 232
Berman, J. K., 419
Bissell, Addison H., 486
Brown, Merle J., 128
Brunn, Harold, 51
Buka, Alfred J., 444
Burrows, Samuel J., 451

Caldwell, J. Hadley, 638
Cantor, Meyer O., 658
Carabba, Victor, 667
Christopher, Frederick, 636
Clute, Howard M., 282
Cole, Warren H., 245
Coller, Frederick A., 1
Collins, Donald C., 454
Collins, Foster K., 454
Crile, George, 123
Crile, George, Jr., 123

DeBakey, Michael, 292
Donaldson, J. K., 505
Drosin, Louis, 580

Emmett, John L., 349
Estes, W. L., Jr., 197

Finney, J. M. T., Jr., 118
Foss, Harold L., 205
Frost, Inglis F., 370

Gale, J. W., 647
Glenn, Frank, 186
Gnassi, A. M., 470
Goldberger, Emanuel, 622
Goldman, Leon, 545
Goldstein, Leopold Z., 529
Gorsch, R. V., 436
Gottesman, J., 645
Graham, Roscoe R., 102
Gray, Howard K., 217
Gurdjian, E. S., 596
Guthrie, Donald, 128

Hamilton, G. Johnson, 432
Hargens, C. W., 626
Hartgraves, T. A., 552
Hermanson, Louis, 223
Hilton, Joseph, 491
Hochman, Samuel, 569
Hodges, Fred Jenner, 146
Holman, Albert W., 365
Horsley, J. Shelton, 30
Howard, T. Leon, 323

Kassebohm, Fred A., 377
Keeley, J. L., 647
Kirklin, B. R., 18
Kruse, Fred H., 178
Krusen, Frank H., 487

Lahey, Frank H., 209
Lazarus, Joseph A., 628
Lynch, Jerome M., 432

Maddock, Walter G., 12
Maliniac, Jacques W., 583
Martin, Albert G., 55
Martz, Harry, 479
Marx, Rudolph, 575
McEvers, Albert E., 662
McGehee, John Lucius, 140
McIntyre, Charles H., 152
McNealy, R. W., 237
Meyer, Nathaniel E., 628
Mider, G. Burroughs, 42
Mixter, Charles G., 223
Mogavero, Francesco, 447
Morgan, E. King, 477
Murray, Samuel, 292

Nitschke, Paul H., 560

Ochsner, Alton, 292
Owen, C. I., 462

Pearl, Felix, 51
Penberthy, Grover C., 232
Pfeiffer, Damon B., 55
Pilcher, Frederick, Jr., 357
Polayes, Silik H., 538
Priestley, James T., 357

Ransom, Henry K., 264
Ravdin, I. S., 171
Reed, Fred Romer, 514
Rentschler, Calvin B., 485
Rife, C. Sherrill, 73

Salzmann, Harry A., 529
Sargent, Willard S., 396
Sauer, Paul K., 475
Schreiber, Milton J., 377
Scott, Wendell G., 157
Scott, W. J. Merle, 42
Shawan, H. K., 70, 462
Sherwin, Benjamin, 413
Silverman, Irving, 671
Siris, Irwin E., 611
Sise, L. F., 22
Smith, Morris, K., 192
Sokoloff, Boris, 673
Stone, Charles M., 477
Sweek, W. O., 552
Sweet, J. E., 162

Van Werden, Benjamin DeKalb, 426
Veal, J. Ross, 426
Voorhees, Irving Wilson, 588

Walters, Waltman, 62
Welch, C. Stuart, 487
Whipple, Allen O., 260
Whitaker, Lester R., 407
Wiener, Morris Frederick, 538
Williams, H. G., 552
Wolfe, Ralph, 613
Wright, W. A., 490

SUBJECT INDEX TO VOLUME XL

(Bo.B.) = Bookshelf Browsing; (E.) = Editorial

Abdomen, surgery of, choice of anesthesia, 22
Abscess
  pyogenic, of liver, 292
  subphrenic, 636
Adrenal cortex
  exhaustion of, 514
  surgical shock and, 514
Allantoin, cod liver oil and, in treatment of suppurative cutaneous wounds, 529
Ampulla, carcinoma of, treatment, 260
Amylase, of blood, in acute pancreatitis, 245
Anesthesia
  in surgery of upper abdomen, 22
  intravenous, in obstetrics, 377
Aponeurosis, use of in reconstruction of internal ring, in indirect inguinal hernia, 618
Appendicitis, perforated, straight pin causing, 485
Appendix, chromargentaffine tumors of, 470

Back, lower, pain in, 444
Bile ducts
  congenital obstruction, 232
  extrahepatic, cancer of, 264
    injuries to, 217
Biliary tract
  diseases, diagnosis, 178
    treatment, 178
  surgery of, in children, 232
Biopsy, new needle for, 671
Bladder
  foreign bodies in, 560
    methods of removal, 560
  multiple calculi of, 477
Bleeding, in peptic ulcer, clinical aspects, 545
Blood, in renal rickets, 325
Bonapartes, cancer in (Bo.B.), 673
Book Reviews:
  Approved Laboratory Technic (Kolmer and Boerner), 502
  Disease and the Man (Lapham), 320
  Elements of Orthopaedic Surgery (Smith), 320
  Introduction to Ophthalmology (Kronfeld), 500
  MacLeod's Physiology in Modern Medicine (Bard, Ed.), 501
  Monograph on Veins (Franklin), 502
  Neurology (Grinker), 320
  Practical Proctology (Buie), 500
  Primary Carcinoma of Lung (Simons), 501
  Science and Practice of Surgery (Romanis and Mitchiner), 501
  Text-Book of Ophthalmic Operations (Grimsdale), 501
  Treatment of Clinical and Laboratory Data (Mainland), 500

Calculi, multiple, of bladder, 477
Cancer
  gastric ulcer and, 42
  of ampullary region, treatment, 260
  of duodenum, 89
  of extrahepatic bile ducts, 264
  of head of pancreas, treatment, 260
  of pancreas, 264
  of rectum, rationale of technique, 432
    surgery in, 432
Cancer, of stomach, 30
    gastrectomy in, 35
  predisposition to, in Bonapartes (Bo.B.), 673
  recurrent intestinal, secondary resection, 645
Carbuncle, 419
Carcinoid tumor, of Meckel's diverticulum, 454
Carotid body, tumors of, 462
Catheterization
  instruments for, 350
  technique, 350
  urethral, difficulties in, 349
Cecum, benign tumors, 538
Cesarean section, transperitoneal exclusion technique, 370
Chart, for seriously sick surgical patient, 626
Children
  biliary surgery in, 232
  cholecystitis in, 233
  jaundice in, 235
Chin
  deformed, rhinoplasty and, 583
  method of reconstruction, 583
Cholangiography
  evaluation, 223
  in subserous cholecystectomy, 552
  results of, 224
Cholecystectomy
  high ligation of cystic vessels in, 407
  partial, in treatment of acute gangrenous cholecystitis, 197
  silver clips in, 407
  subserous, 407
    cholangiography in, 552
    cystic duct drainage in, 552
Cholecystitis
  acute, early surgical treatment, 186
    gangrenous, treatment, 197
    treatment, 192
  cholecystography in, 146
  in children, 233
Cholecystography
  in cholecystitis and cholelithiasis, 146
  in gall-bladder surgery, 146
  interpretation of faint shadows in, 157
  iso-iodeikon in, 152
  liver function determination and, 152
  value of, 146
Cholelithiasis, cholecystography in, 147
Chorionepithelioma, 638
Chromargentaffine tumors, of appendix, 470
Cleft palate, operations for, causes of failure, 588
Cod liver oil, allantoin and, in treatment of suppurative cutaneous wounds, 529
Colon, fistula, 73
Colostomy stoma, applicator for Elliott treatment through, 487
Common duct
  indications for exploration, 212
  obstruction of, 396
    causes, 396
    diagnosis, 398
    symptoms and signs, 397
    treatment, 400
  stones in, 209
Congratulations, to W. B. Saunders (E.), 322
Cranium, internal hemorrhage, operative management 596
Cystic duct, drainage, in subserous cholecystectomy, 552

Cysts
enterogenous, 413
mesenteric, causing intestinal obstruction, 647
pericecal, 413

Diagnosis, surgical, gastroscopy in, 5
Digestion, surgery of (E.), 1
Dignity in medicine (E.), 321
Diverticula
of duodenum, 128
of jejunum, 128
Duodenum
carcinoma of, 89
chronic obstruction, 140
dilatation of, 140
diseases of, nutritional disturbances and, 12
diverticula of, 128
ulcer of, indications for operation in, 102, 123
surgery in, 102
results, 123
treatment, 102
Dwarfism, renal, 323

Ectopic pregnancy, simulating intestinal obstruction, 479
Editorials:
Congratulations and Best Wishes, 322
Development of Surgery of the Upper Digestive Tract, 1
Dignity in Medicine, 321
Organized Medicine, 503
Electrosurgery
currents, 436
in proctology, 436
Elliott treatment, applicator for, through colostomy stoma, 487
Evipal soluble, in obstetrics, 377

Fascia, aponeurotic, in reconstruction of internal ring, in indirect inguinal hernia, 618
Femoral hernia, repair of, 451
Fibroma
mesenteric, of terminal ileum, 539
perirenal, 628
Fistula
bronchial, in subphrenic abscess, 636
gastrojejunocolic, 73
Fracture
of hip, 447
spinal, method of transportation, 611

Gall-bladder
diseases, diagnosis, 178
indications for operation in, 205
treatment, 178
faint shadows, interpretation, 157
surgery of, cholecystography in, 146
complications, 282
Gallstones, formation of, 162
Gastrectomy, in stomach cancer, 35
Gastritis, gastroscopy in, 7
Gastroduodenostomy, physiology, 118
Gastroenterostomy, jejunal ulcer following, 475
Gastrojejunocolic fistula, 73
Gastrojejunostomy, physiology, 118
Gastroscopy
in gastritis, 7
in surgical diagnosis, 5
in tumors, 5
in ulcer, 6
method of, 5
Graft, dermal, in reconstruction of chin, 583
Gynecology, urinary tract and, 569

Hemorrhage, traumatic intracranial, operative management, 596
Hepatic duct, stones in, 209
Hernia
femoral, repair of, 451
indirect inguinal, reconstruction of internal ring in, 618
management of sac in, 613
Herniorrhaphy, femoral, 451
Hip, fractures of, 447

Ileum
benign tumors, 538
mesenteric fibroma of, 539
Intestine
cysts in, 413
Elliott treatment of, through colostomy stoma, 487
obstruction of, 505
complicating thyrotoxicosis, 662
ectopic pregnancy simulating, 479
mesenteric cysts causing, 647
secondary resection, in recurrent carcinoma, 645
Iso-iodeikon
in cholecystography, 152
in liver function determination, 152

Jaundice
in children, 235
preoperative care, 237
Jejunum
diverticula of, 128
fistula, 73
ulcer of, after gastroenterostomy, 475

Kidney
distortion, perirenal tumors and, 630
function, pregnancy and, 365
traumatic lesions, 357
tumors, causing renal distortion, 630

Lipoma, of ileocecal valve, 538
Liver, function, 171
determination of, cholecystography and, 152
iso-iodeikon in, 152
normal, 171
pathologic, 171
pyogenic abscess, 292

Maggot therapy, active principle, 529
Marriage, unequal (Bo.B.), 491
Meckel's diverticulum, ulcerating carcinoid tumor of, 454
Medical societies (E.), 503
Medicine, dignity in (E.), 321
Medicine, organized (E.), 503
Mesentery, cysts of, causing intestinal obstruction, 647
Monoethanolamine oleate, for injection of varicose veins, 628

Needle, for biopsies, 671
Nutrition, gastro-intestinal diseases and, 12

Obstetrics
evipal soluble in, 377
intravenous anesthesia in, 377
pentothal in, 377

Obstruction, intestinal, 505
complicating thyrotoxicosis, 662
ectopic pregnancy simulating, 479
mesenteric cysts causing, 647

Pain, in lower back, 444
Pancreas
cancer of, 264
head of, cancer of, treatment, 260
Pancreatitis, acute, 245
blood amylase test in, 245
pathogenesis, 245
Patella
non-recurrent dislocation, 654
total removal of, 486
Pellegrini-Stieda's disease, 658
Pentothal, in obstetrics, 377
Peptic ulcer
bleeding, clinical aspects, 545
surgical treatment, 55
perforation of, 70
Pin, causing perforated appendicitis, 485
Polyposis, gastric, 51
Pregnancy
ectopic, simulating intestinal obstruction, 479
measurement of kidney function in, 365
Proctology, electrosurgery in, 436
Protein deficiency, gastrointestinal disease and, 15

Rectum
carcinoma of, rationale of technique, 432
surgery in, 432
Renal rickets, 323
Rhinoplasty, chin reconstruction and, 583
Rickets, renal, 323

Sac, hernial, management of, 613
Salpingitis, non-specific, 575
Saphenous vein
ligation of, in treatment of varicose veins, 426
physiologic basis, 426
Saunders, congratulations to (E.), 322
Sex development, in renal rickets, 327
Shock
surgical, adrenal cortex exhaustion and, 514
causes, 515
Sigmoid, carcinoma of, rationale of surgical technique, 432
Silver clips, in cholecystectomy, 407
Skin, suppurative wounds of, treatment with cod liver oil and allantoin, 529
Spine, fractures of, method of transportation, 611
Square knot
one-handed thumb-roll maneuver, 622
rationale of technique, 622
Stomach
cancer of, 30
gastrectomy in, 35
diseases of, nutritional disturbances and, 12
fistula, 73
multiple polyposis of, 51
surgery of, complications, 282
ulcer, chronic, 62
malignancy in, 42
indication for operation in, 62
perforation, 70
Stones
in common duct, 209
in hepatic duct, 209
Sturmdorf tracheloplasty, modified, 580
Suction apparatus, 667
Suction siphonage apparatus, 490
Suppuration, in cutaneous wounds, treatment with cod liver oil and allantoin, 529
Surgery
in bleeding peptic ulcer, 55
in duodenal ulcer, results of, 123
in gall-bladder disease, indications, 205
in jaundiced patients, 237
in syringomyelia, 384
of gall-bladder, cholecystography in, 146
complications, 282
of stomach, complications, 282
of upper digestive tract (E.), 1
upper abdominal, anesthesia in, 22
Surgical patient, seriously sick, chart for, 626
Syringomyelia, surgical treatment, 384

Thyrotoxicosis, acute intestinal obstruction in, 662
Tracheloplasty, modified Sturmdorf, 580
Transportation, in spinal fractures, 611
Trauma
causing intracranial hemorrhage, 596
to kidney, 357
Tumors
benign, of ileocecal region, 538
carcinoid, of Meckel's diverticulum, 454
chromargentaffine, of appendix, 470
gastroscopy in, 5
of carotid body, 462
perirenal, causing renal distortion, 630

Ulcer
duodenal, indications for operation in, 102, 123
gastric, chronic, 62
indications for operation, 62
malignancy in, 42
gastroscopy in, 6
jejunal, after gastroenterostomy, 475
peptic, bleeding, clinical aspects, 545
surgical indications, 545
surgical treatment, 55
Ulcer, peptic, perforation of, 70
treatment, 70
x-ray diagnosis, 18
Unequal marriage (Bo.B.), 491
Urethra
anatomy, 349
catheterization, difficulties in, 349
Urinary tract symptoms, in gynecology, 569
Urology, in gynecologic service, 576

Varicose veins
injection with monoethanolamine oleate, 628
ligation of saphenous vein in, 426
Vitamin deficiencies, gastrointestinal disease and, 12

Wounds, suppurative cutaneous, treatment with cod liver oil and allantoin, 529

X-ray, in diagnosis of peptic ulcer, 18